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OBSERVER ERROR AND VARIATION IN THE INTERPRETATION OF ELECTROCARDIOGRAMS IN AN EPIDEMIOLOGICAL STUDY OF CORONARY HEART DISEASE

BY

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It is widely accepted that the ECG is one of the most useful devices in the diagnosis of coronary heart disease, but it is important to establish whether, under the conditions of epidemiological research, the normal and abnormal ECG can be reliably and repeatedly distinguished from each other. The problem is in effect one of assessing the performance of an observer, and in doing this it is useful to differentiate between observer error and observer variation.

These two factors have been clearly distinguished and defined by Fletcher and Oldham (1959). "It is appropriate", they write, "to speak of observer error when, in the case of a simple classification into two categories of normal and abnormal, an observer places a case into one group and it can be shown, either by a majority opinion of a number of observers [or] by a subsequent re-examination by the same observer, . . . that the case really belongs to the other group." They continue, in their definition of observer *variation*, "In the alternative and more frequent situation of a continuum where there are two or more categories and the observer is attempting to place a given case into the correct one, disagreement between observers, or in unrepeatability of the same observer may not stem entirely from error. . . . Subjects may be expected to fall on any part of the continuum, although possibly with greater frequency at one end or the other, and those near a boundary will inevitably be misclassified, more and more frequently the nearer they are to it, if there is any source of uncertainty at all."

By publishing a series of standard electrocardiographic tracings, the W.H.O. (1959)† attempted to define what does, and what does not constitute coronary heart disease. This does not, however, do away with observer variation, because in many cases opinions will differ as to whether the changes illustrated in the monograph are in fact present.

In a recent survey of the heart condition of a population sample‡ of over 220 elderly men—a preliminary report of which has already appeared (Acheson and Acheson, 1958)—the ECGs were all read by a single assessor. In order that some idea may be gained of how the frequency of heart disease (and in particular of coronary heart disease) in this sample compares with that in other samples or surveys, it is important that an estimate should be made of this assessor's observer *error*. In addition, it is of value in the final analysis of such a survey to know whether any pattern in an ECG leads to observer *variation*; whether, in Fletcher and Oldham's words, a specific pattern or patterns are "near the borderline".

Material and Method

The men were aged 65 to 85 and had been employed by a large Dublin industrial concern; full details of the selection of the sample and ECG technique are published elsewhere (Acheson and Acheson, 1958; Acheson, Hemmens, and Jessop, 1958). The tracings were made with a direct writing machine, and each tracing consisted of leads I, II, III, aVL, aVR, aVF, V1, V2, V3, V4, V5 and V6. Since it was impracticable to have all the tracings assessed by a number of different readers, it was decided to use a 25 per cent. sample. The cases were numbered, and the sample was chosen by taking a random number between 1 and 165—it proved to be 156—and then using 56 consecutive tracings numbered from 156 inclusive. They were all assessed from the intact original tracing by one observer (Assessment A_1); then the author, who made no

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† The electrocardiographic tracings, which are published as an annex to this otherwise excellent monograph, would appear to have been selected from clinical rather than epidemiological experience and are unlikely to prove helpful in survey work.

‡ "Population Sample" in this series of studies has been used in the sense of an age-stratified random sample from a population of employees of a large industrial concern, so that the sample may be distinguished from those drawn entirely from old people's homes, hospitals, or geriatric clinics. No claim is made that the sample in question is representative of the old men of Dublin.

assessments, cut the tracings up and mounted them on cards, so that two or three complexes were shown for each lead (Fig. 1). Then three other observers (Assessments B, C, and D) read the tracings, and finally the first observer read them again (Assessment A_2). Thus, although four assessments were

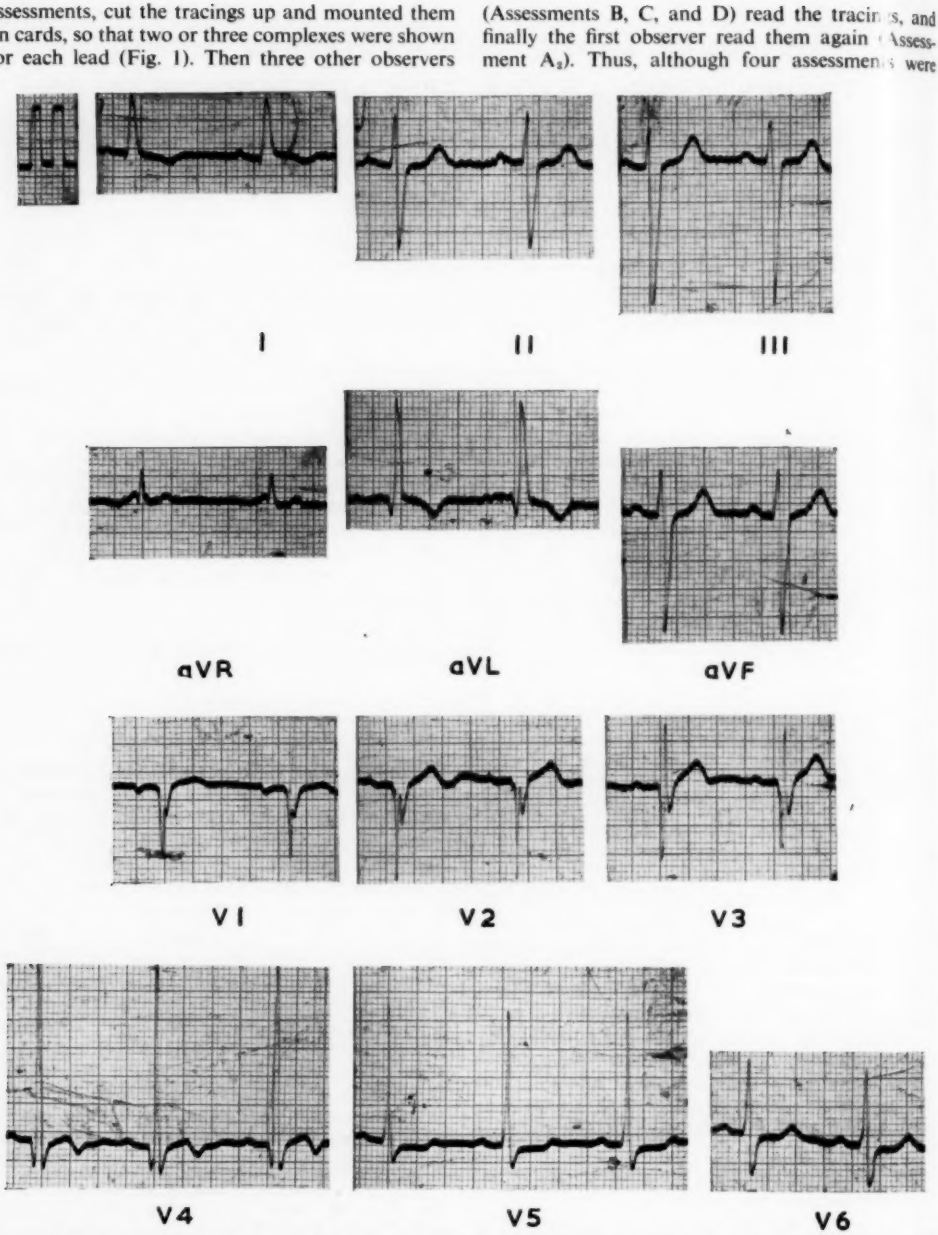

FIG. 1.—Specimen showing the form in which the ECGs were presented for all assessments (except in A_1 which was made from the intact tracing). The case shown is 213, the only one in which it was agreed in all assessments that the changes were diagnostic of Myocardial Infarction. Two observers felt that Left Ventricular Hypertrophy was also present.

made from the same mounted ECGs, Observer A's first assessment was made from more complete material; the possible significance of this unavoidable difference will be discussed further below. Of the four assessors, all of whom are members of the Royal College of Physicians, three (including Observer A), had special experience in electrocardiography, and the fourth was an epidemiologist particularly interested in diseases of the cardiovascular system.

Each of the observers was asked to fill in a prescribed form in respect of each tracing (Fig. 2). All the assessments were done independently and, with the exception of two who are acquainted, no observer was personally known to the others. The second occasion on which Observer A was asked to read the 56 tracings was more than 12 months after the first, and he had no record of his original interpretations. Furthermore, at the time of making his first interpretations he did not know that he would

GUINNESS' PENSIONERS SURVEY

INTERPRETATION OF ELECTROCARDIOGRAPHS

OBSERVER'S NAME OR CODE .....

CASE NO..... NAME..... AGE..... B.P...../.....

NUMBER OF TRACINGS [1] [2] DIGITALIS THERAPY [Yes] [No] PULSE RATE [ ]

1. **P WAVE**
   Normal
   Other (specify).....
2. **P.R. INTERVAL** (from Lead II)
   Normal
   Other (specify).....
3. **QRS COMPLEX**
   (a) QRS Interval (measured from Lead V_2).....
   (b) Axis Deviation: Nil Left Right
   (c) Bundle Branch Block: Nil Left Complete
   Right Incomplete
4. **MYOCARDIAL INFARCTION**
   Q Waves
   Suggestive of Myocardial Infarction (specify leads).....
   Indicative of Myocardial Infarction (specify leads).....
5. **RS-T SEGMENT** (excluding Myocardial Infarction)
   Abnormally Elevated (specify leads).....
   Normal
   Abnormally Depressed (specify leads).....
6. **T-WAVE** (excluding Myocardial Infarction)
   Normal
   Absent (specify leads).....
   Inverted (specify leads).....
7. **DYSRHYTHMIAS**
   (a) None
   (b) Atrial Fibrillation
   (c) Tachycardia: Paroxysmal Supraventricular
   Ventricular
   (d) Bradycardia
   (e) Ectopic Beats: Supraventricular
   Ventricular
   Interpolated
   Multifocal
8. **DIAGNOSIS OF ELECTROCARDIOGRAPHIC TRACING AT REST**

PLEASE COMPLETE FORM BY RINGING APPROPRIATE WORD WHERE POSSIBLE

FIG. 2.—Prescribed form which the four observers were required to complete when they had interpreted the ECGs.

be asked to read the tracings again.

The only information the observers were given about the old men from whom the tracings were made was age, blood pressure, and whether or not the subject had been taking digitalis—none of the sub-sample of 56 was, in fact, taking this drug.

Results

Comparison between the First and Second Assessments of Observer A

Observer A considered three of the 56 tracings to be unsuitable, either because they were incomplete or because they were technically imperfect and these were therefore discarded; on his first reading he classed 27 of the remaining 53 as normal (Group I), eight as having possible abnormalities (Group II), and eighteen as definitely abnormal (Group III). On his second reading he placed fewer tracings in Group II, with a consequent increase in the sizes of Groups I and III (Table I).

Table I

Classification of 53 ECGs into three main groups by observer A

Assessment		A_1	A_2
Group	I. Normal	27	31
	II. ? Abnormal	8	2
	III. Abnormal	18	20
Total		53	53

Table II gives a more detailed analysis of the placing of the tracings in the three groups at the two assessments. It will be seen that, apart from one tracing classed as normal at A_1 and abnormal at A_2, and another moved from Group III to Group II at the second reading, all the differences are concerned with the eight tracings placed in Group II at Assessment A_1.

Table II

Grouping at A_2 of assessments made at A_1

Grouping at A_1	Grouping at A_2			Total
	I	II	III	
I	26	0	1	27
II	5	1	2	8
III	0	1	17	18
Total	31	2	20	53

Comparison between the Assessments of Observer A and those of Observers B, C, and D

The fairest way of comparing the performance of Observer A with those of B, C, and D is in terms of Assessment A_2, because this was carried out from mounted ECGs, whereas Assessment A_1 had been carried out from the original unmounted strips. Table III shows how the grouping of the other observers compared with that of A_2; by reading this Table from the top left corner diagonally to the bottom right corner, it is evident that there was agreement with Assessment A_2 on 134 (84 per cent.) occasions.

Table III

Comparison between grouping at A_2 and other assessments of mounted ECGs

Grouping at A_2	Grouping at B, C, and D			Total
	I	II	III	
I	84	3	6	93
II	2	2	2	6
III	6	6	48	60
Total	92	11	56	159

In thirteen (8 per cent.) assessments an ECG was placed in the group next to that designated at Assessment A_2; these are called first degree disagreements. Twelve (8 per cent.) of the subsequent assessments were second degree disagreements, that is, a tracing classified as Group I at A_2 was classified subsequently as Group III or *vice versa*. This analysis is shown in Table IV, which also gives a similar summary of the differences shown between A_1 and A_2 in Table II, and of all subsequent assessments compared with A_1.

Table IV

Degree of disagreement in grouping A_2 with A_1, B, C, and D with A_2 and all subsequent assessments with A_1

Comparison		A_2 with A_1		B, C, and D with A_2		A_2, B, C, and D with A_1	
		No.	Per cent.	No.	Per cent.	No.	Per cent.
Agreement		44	83	134	84	163	77
Disagreement	First Degree	8	15	13	8	42	20
	Second Degree	1	2	12	8	7	3
Totals		53	100	159	100	212	100

To summarize so far, when Observer A is tested for his ability to repeat his own performance, the most striking difference between his two sets of assessments is that the second is more positive, inasmuch as it has fewer Group II classifications than the first. This may be because the process of mounting the tracings left him with selected complexes to study when he made his second series of assessments, a suggestion which is to some extent borne out by the fact that the frequency of Group II classifications

in assessments B, C, and D is more similar to the pattern of A_2 than any of the four are to that of A_1. The effect of this increased positivity, however, is to make the number of second degree disagreements higher when A_2 is compared with the other three observers than when it is compared with A_1, or when A_1 is compared with all four assessments (Table IV).

The number of disagreements with the actual diagnosis as opposed to the grouping reached at Assessment A_1 are shown in Table V, where it can be seen that there was some measure of disagreement in respect of 21 tracings, six of which were originally placed in Group I, eight in Group II, and seven in Group III. Three of the five in Group II which were considered to be normal at Assessment A_2 were also grouped as normal at all subsequent assessments; because all observers were unanimous about these three ECGs in their mounted form, they will not be considered further, nor will three placed in Group I at Assessment A_1, but grouped as "? abnormal" by just one other observer. The remaining fifteen of the 21 ECGs were subject to a wider margin of disagreement and are therefore worthy of a more detailed discussion.

TABLE V

OCCASIONS ON WHICH ASSESSMENTS A_2, B, C, AND D DISAGREED WITH THE PRECISE DIAGNOSIS MADE AT ASSESSMENT A_1

Group	Number of Disagreements on Each ECG					
	None	One	Two	Three	Four	Totals
I	21	5	1	—	—	27
II	—	2†	2	1	3	8
III	11	—	2*	3*	2*	18
Total ECGs . .	32	7	5	4	5	53

* One case in each group marked thus was diagnosed by A_1 "Left Ventricular Hypertrophy *or* Myocardial Ischaemia".
† One case in this group was diagnosed by A_1 "? Left Ventricular Hypertrophy ? Myocardial Ischaemia".

FURTHER DESCRIPTION OF ECGS GIVING RISE TO A WIDE DIFFERENCE OF OPINION

In this section the fifteen "difficult" ECGs, together with the diagnoses at all five assessments, will be shown; for the sake of simplicity the grouping made at Assessment A_1 will be used as a basis for classification.

Group I.—The three tracings giving rise to definite disagreement in Group I are shown (Fig. 3, overleaf, pp. 104–106) together with the diagnoses offered at all assessments (Table VI). It will be seen that only one observer doubted the normality of two of the tracings—but that two considered the third tracing (189) abnormal, one of these being Observer A himself. It is worth noting that the suggested abnormalities were Left Ventricular Hypertrophy (on three occasions) and Infarct (once).

Group II.—The eight cases in which Observer A was uncertain whether the tracing was abnormal at his first reading were those about which the difference of opinion was greatest. In three (two ? Infarction and one ? Left Ventricular Hypertrophy), the possibility of abnormality was not raised in any of the four subsequent assessments. The other five cases are shown in Fig. 4 (overleaf, pp. 107–111), and the various interpretations are given in Table VII (overleaf, p. 112). One case (175) presents a special difficulty because the subject suffered from a gross Parkinsonian muscular tremor. In the others the chief difficulty lay in deciding whether the tracing was in fact normal (158, 190, 215) and in making a distinction between Left Ventricular Hypertrophy and Myocardial Ischaemia (190 and 193).

Group III.—Eighteen tracings were declared abnormal by Observer A on his first reading, and in eleven there was a wide measure of agreement as to the nature of the abnormality. These were diagnosed by him as Left Ventricular Hypertrophy (five), Myocardial Ischaemia (one), Myocardial Ischaemia *or* Left Ventricular Hypertrophy (one), Atrial

TABLE VI*

INTERPRETATIONS OF THREE GROUP I ECGS ABOUT WHICH THERE WAS SECOND DEGREE DISAGREEMENT AS TO GROUPING (SEE FIG. 3)

Case No.	Age (yrs)	Blood Pressure		Interpretations of Observers				
		Systolic	Diastolic	A_1	A_2	B	C	D
167	72	150	85	Normal	Normal	Left Ventricular Hypertrophy	Normal	Normal
189	72	210	120	Normal	Old Infarct	Normal	Left Ventricular Hypertrophy	Normal
207	70	180	105	Normal	Normal	Normal	Left Ventricular Hypertrophy	Normal

* In Tables VI to IX inclusive the observer's original wording has been used wherever possible.

CASE 167

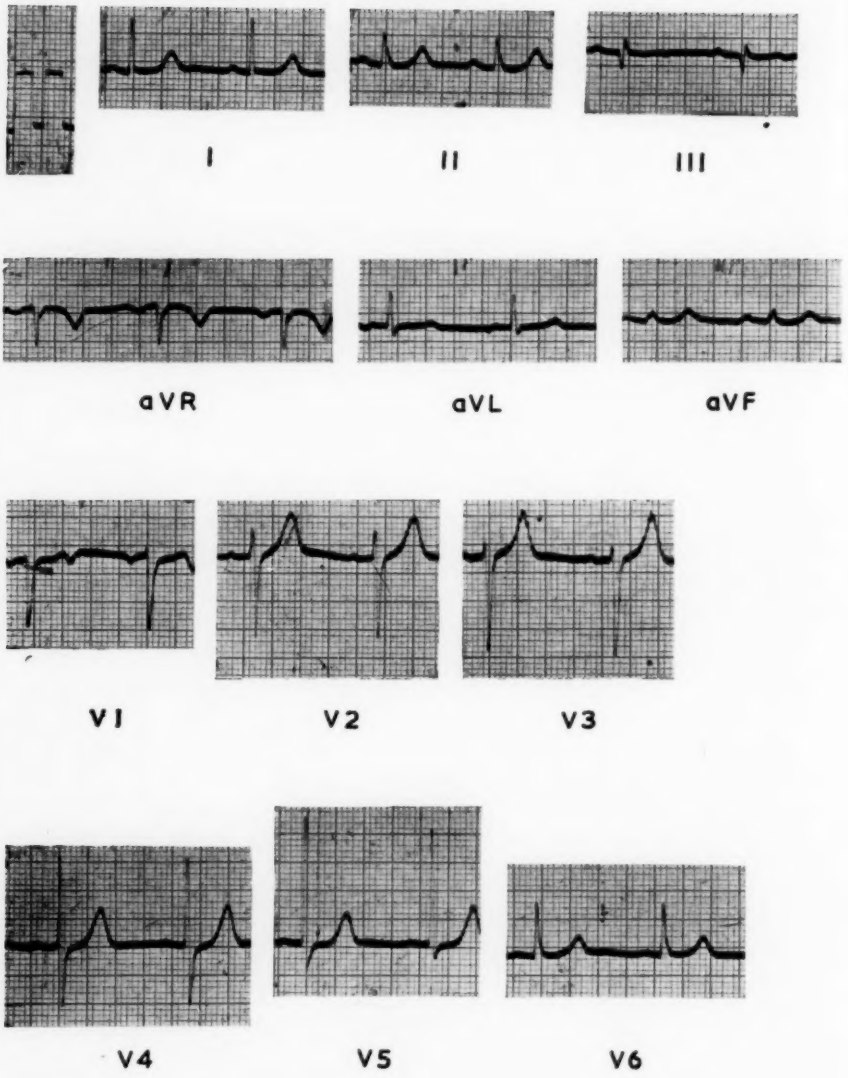

FIG. 3.—Three ECGs which were placed in Group I at Assessment A_1 and gave rise to second degree disagreement in grouping (see also Table VI).

CASE 189

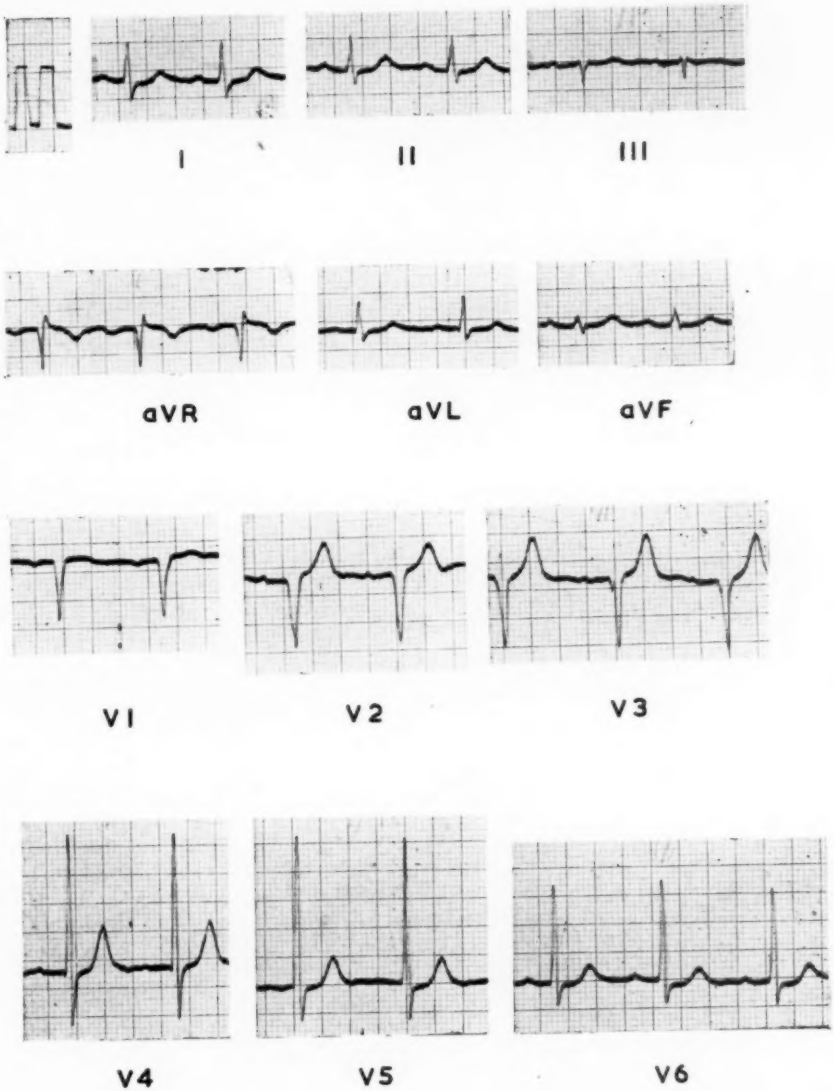

FIG. 3 (*continued*).

CASE 207

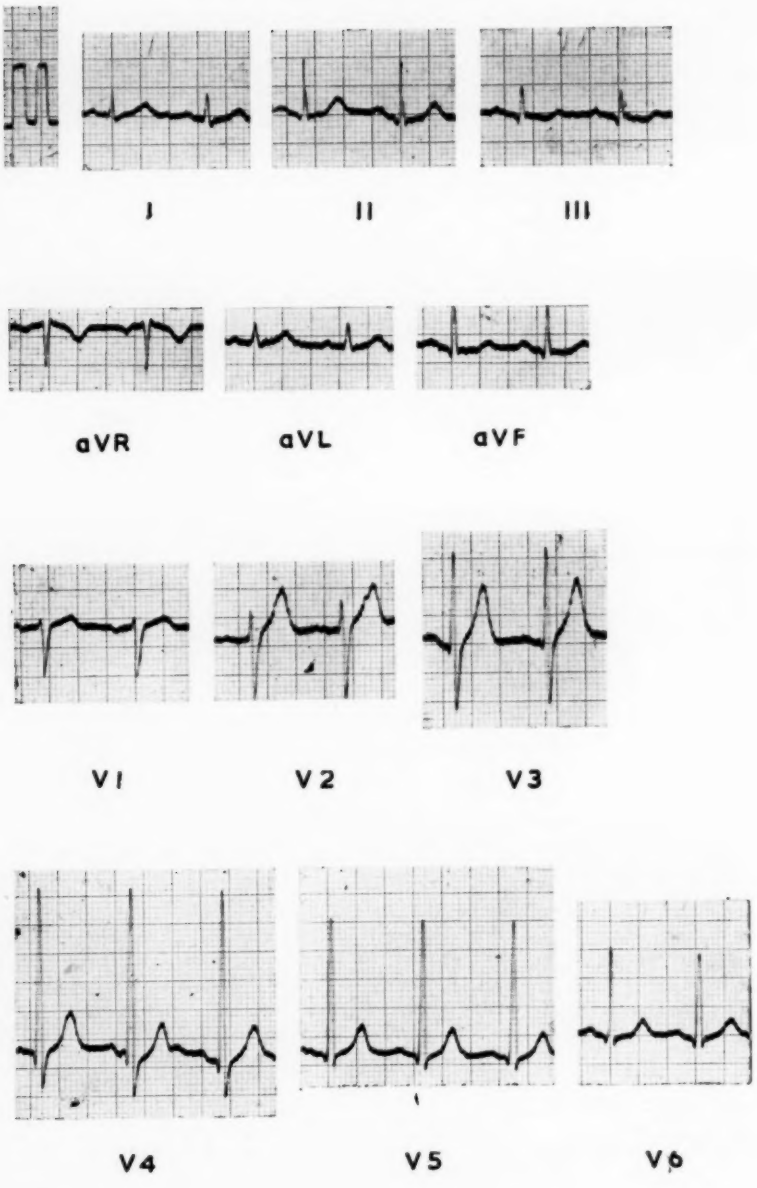

FIG. 3 (*continued*).

CASE 158

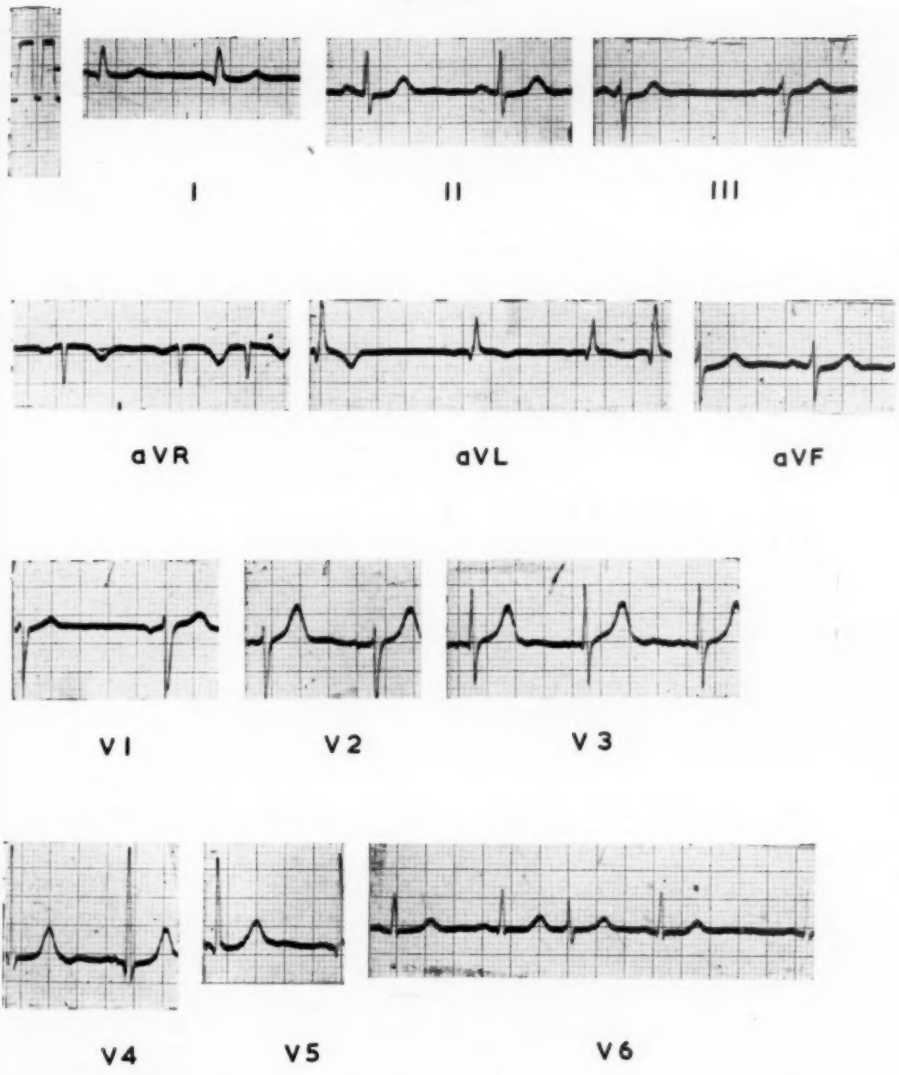

FIG. 4.—Five ECGs in Group II which gave rise to disagreement in grouping (see also Table VII).

CASE 175

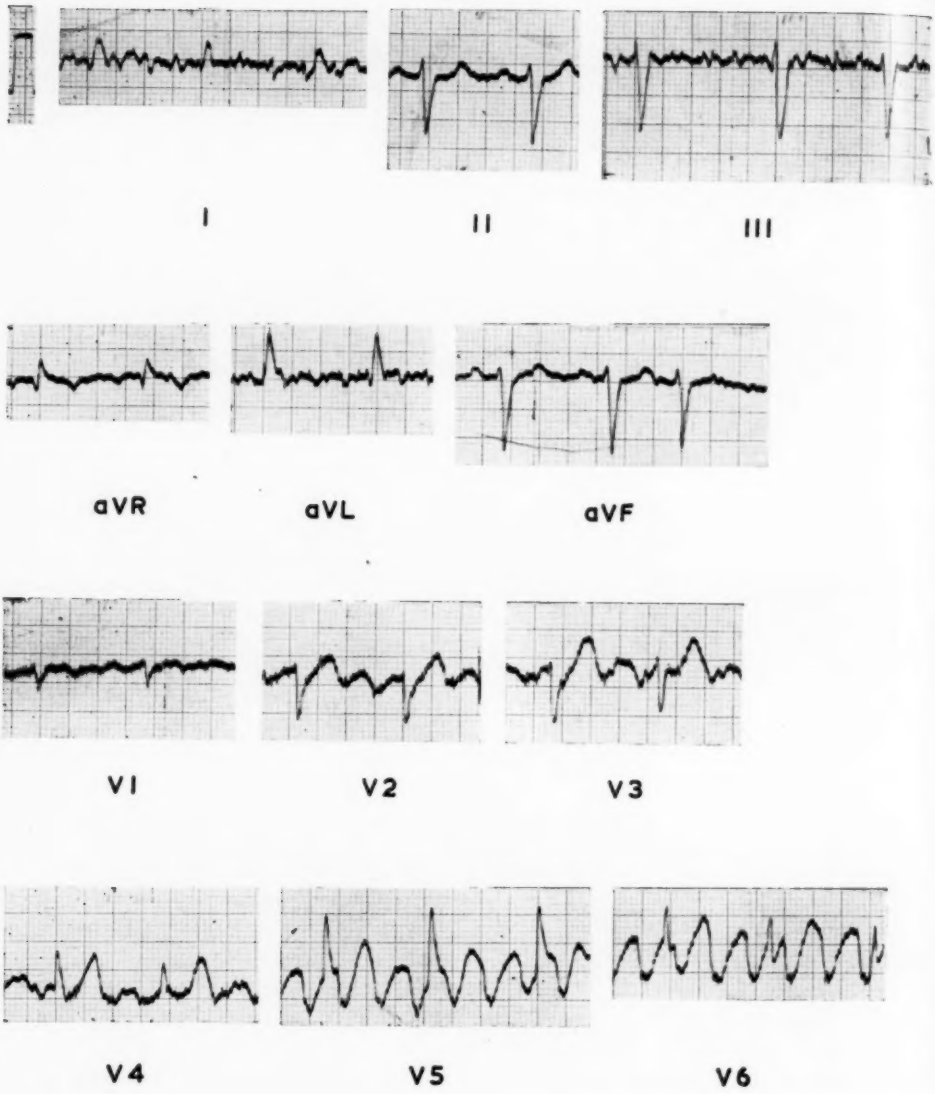

FIG. 4 (*continued*).

CASE 190

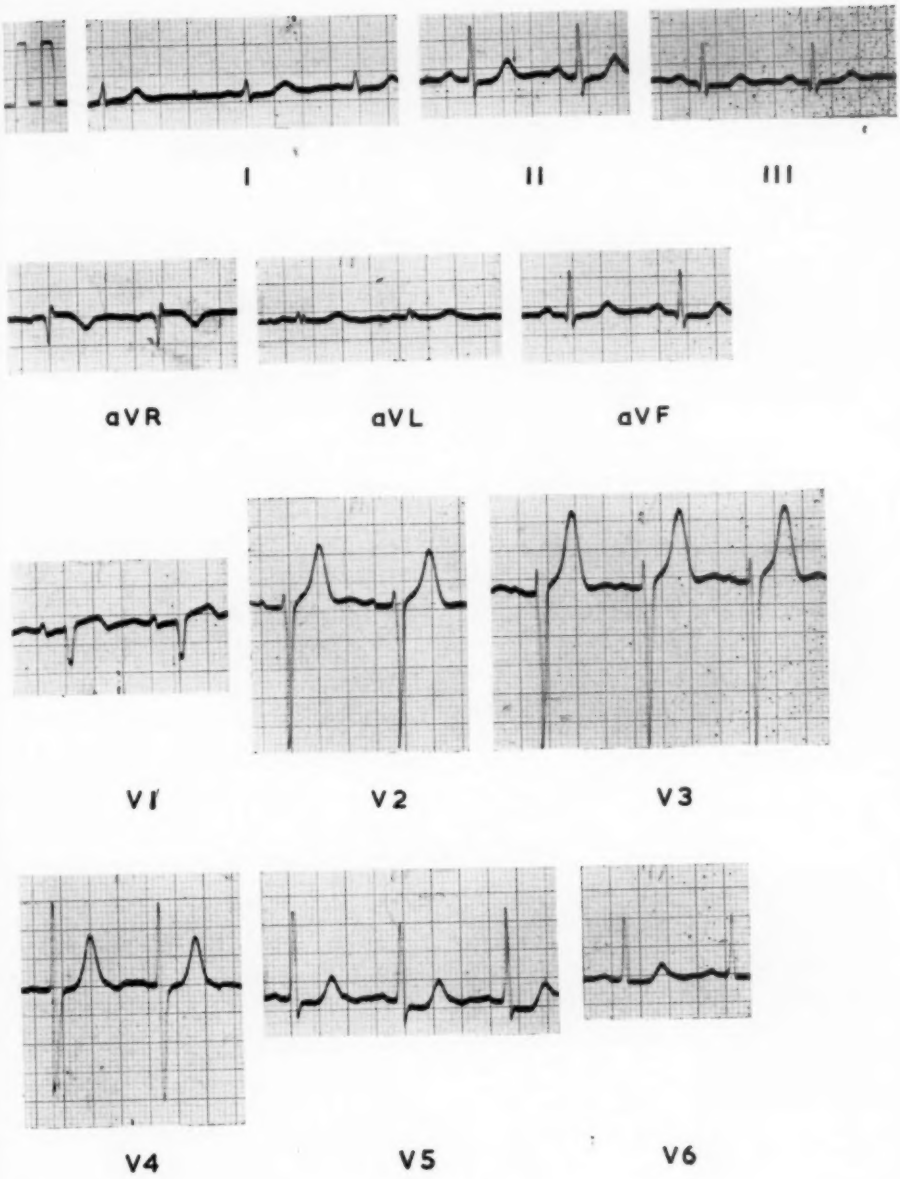

FIG. 4 (*continued*).

CASE 193

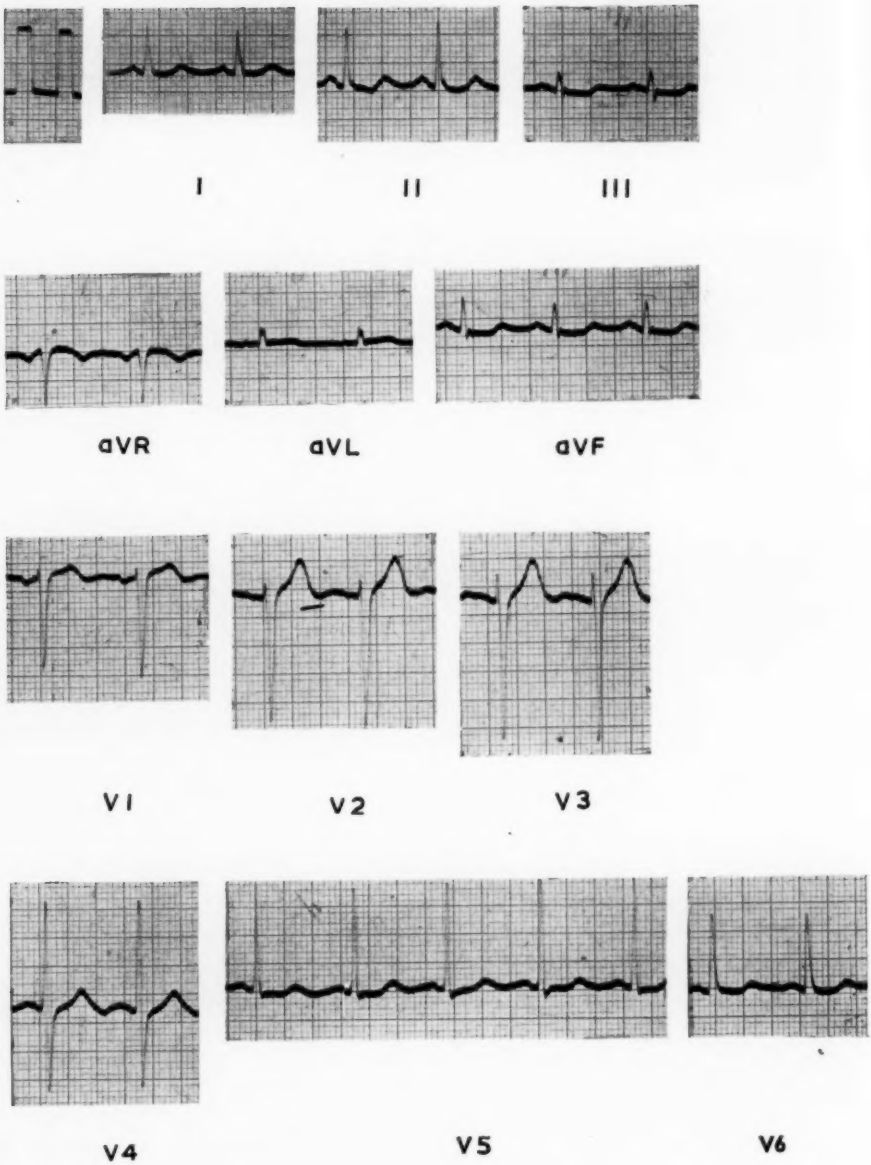

FIG. 4 (*continued*).

CASE 215

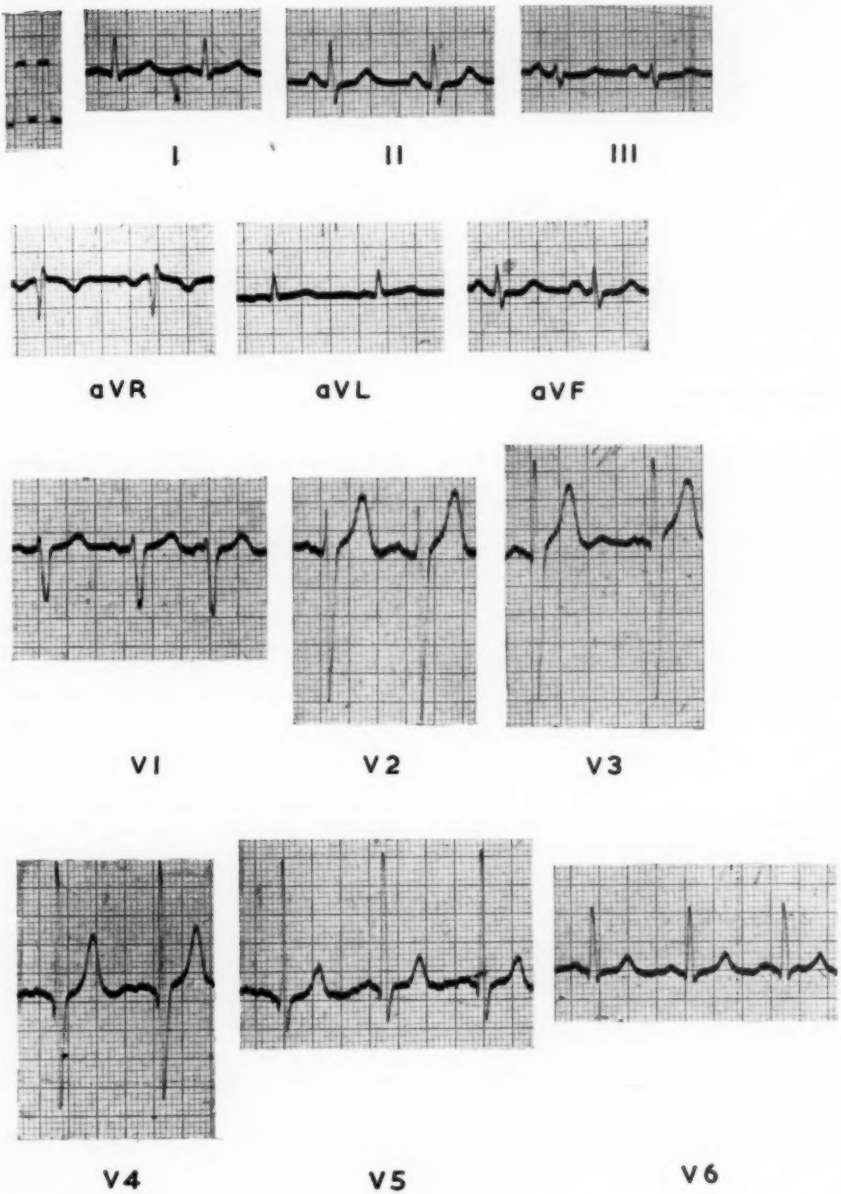

FIG. 4 (*continued*).

TABLE VII

INTERPRETATIONS OF FIVE GROUP II ECGs (SEE FIG. 4)

Case No.	Age (yrs)	Blood Pressure		Interpretations of Observers				
		Systolic	Diastolic	A_1	A_2	B	C	D
158	73	150	75	? High Anterior Infarct	? Small Infarct	Probable Infarction	Old Anterior Infarct	Normal
175*	72	140	85	? Bundle Branch Block (could be normal)	Normal	Left Ventricular Hypertrophy	Left Bundle Branch Block	? Normal
190	71	210	90	? Myocardial Ischaemia ? Left Ventricular Hypertrophy	Myocardial Ischaemia	Normal	Left Ventricular Hypertrophy (could be Ischaemia)	? Myocardial Ischaemia
193	71	175	110	? Left Ventricular Hypertrophy	Myocardial Ischaemia (could be Left Ventricular Hypertrophy)	Myocardial Ischaemia	Left Ventricular Hypertrophy (could be Myocardial Ischaemia)	Myocardial Ischaemia
215	69	240	130	? Left Ventricular Hypertrophy	Normal	Left Ventricular Hypertrophy	Left Ventricular Hypertrophy	Normal

* Case 175 had a severe Parkinsonian tremor (see Fig. 4).

Fibrillation (three), and Myocardial Infarction (one). In four of the five cases with Left Ventricular Hypertrophy, this was considered to be the most likely diagnosis at all the four succeeding assessments; in the fifth case, Ischaemia was put forward as more likely at all the four later assessments. In the case considered at Assessment A_1 to be "Myocardial Ischaemia *or* Left Ventricular Hypertrophy", the same diagnosis was made at A_2; at B and C the diagnosis was "Ischaemia", and at D it was "Ischaemia ? Infarction". Similarly, in the single case of Myocardial Ischaemia, the possible alternative diagnosis of "? Left Ventricular Hypertrophy" put forward by Observer A at his first assessment, was repeated at A_2 and also put forward by Observer B.

All the observers agreed that there was Atrial Fibrillation in three cases, and that this was uncomplicated in one of the three. It was agreed by all that there was a further complication in the other two cases; in Case 185 this was thought to be Left Ventricular Hypertrophy or Myocardial Ischaemia, and in Case 159 an Infarct by A (both assessments) and C, or Ischaemia by B and D. In the one case (213) in which all observers agreed on the diagnosis of Myocardial Infarction, A_1, B, and C thought that Left Ventricular Hypertrophy was also present (Fig. 1).

In four cases opinions differed considerably as to diagnosis; the ECGs of these cases are shown in Fig. 5 (opposite, pp. 113–116), and the interpretation in Table VIII. In all of them the original diagnosis (A_1) was Left Ventricular Hypertrophy; in two cases this was also suggested by another observer, and in all cases Myocardial Ischaemia and Myocardial Infarction were offered as alternatives.

TABLE VIII

INTERPRETATIONS OF FOUR ECGs WHICH NO OBSERVER CONSIDERED NORMAL BUT ABOUT WHICH THERE WAS DISAGREEMENT AS TO DIAGNOSIS (SEE FIG. 5)

Case No.	Age (yrs)	Blood Pressure		Interpretations of Observers				
		Systolic	Diastolic	A_1	A_2	B	C	D
157	70	170	105	Left Ventricular Hypertrophy	Left Ventricular Hypertrophy	Probable Infarct	Ischaemia	? Ischaemia
181	78	150	95	Left Ventricular Hypertrophy	? Ischaemia	Left Ventricular Hypertrophy ? Infarct	Old Anterior Infarct	? Ischaemia
183	71	175	85	Left Ventricular Hypertrophy	Ischaemia ? Old Infarct	Old Infarct	Ischaemia	Ischaemia ? Old Infarct
209	69	170	100	Left Ventricular Hypertrophy	Left Ventricular Hypertrophy *or* Ischaemia	Gross Left Ventricular Hypertrophy	Anterior Infarct *or* Ischaemia	Ischaemia

CASE 157

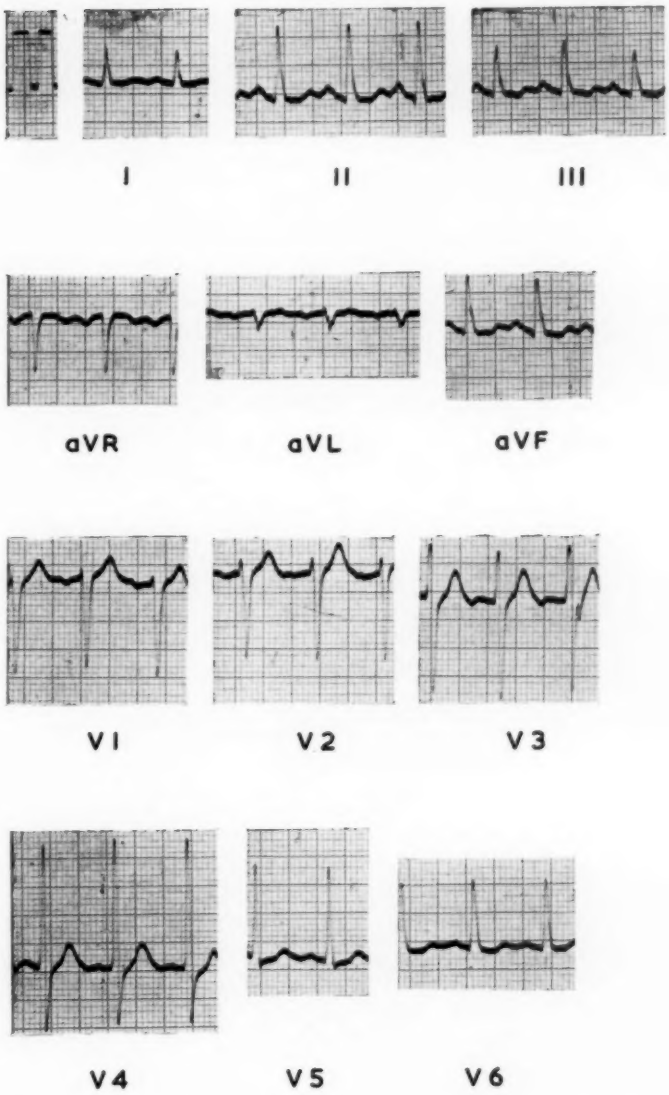

FIG. 5.—Four ECGs in Group III about which there was disagreement in precise diagnosis (see also Table VIII).

CASE 181

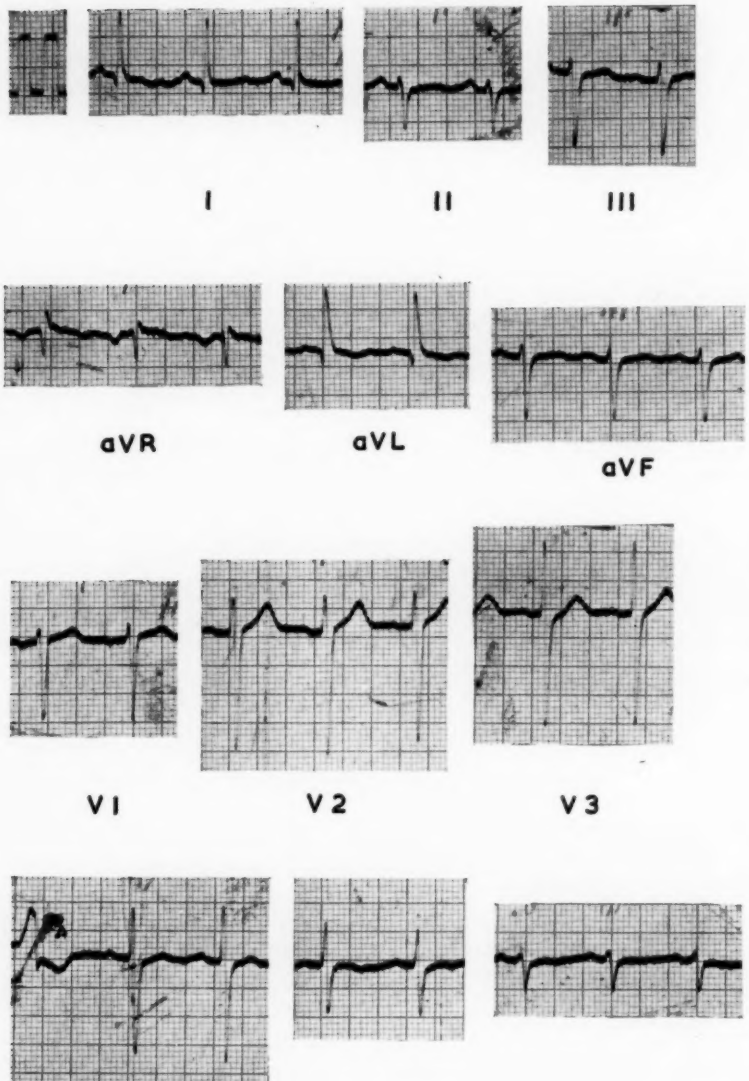

FIG. 5 (*continued*).

CASE 183

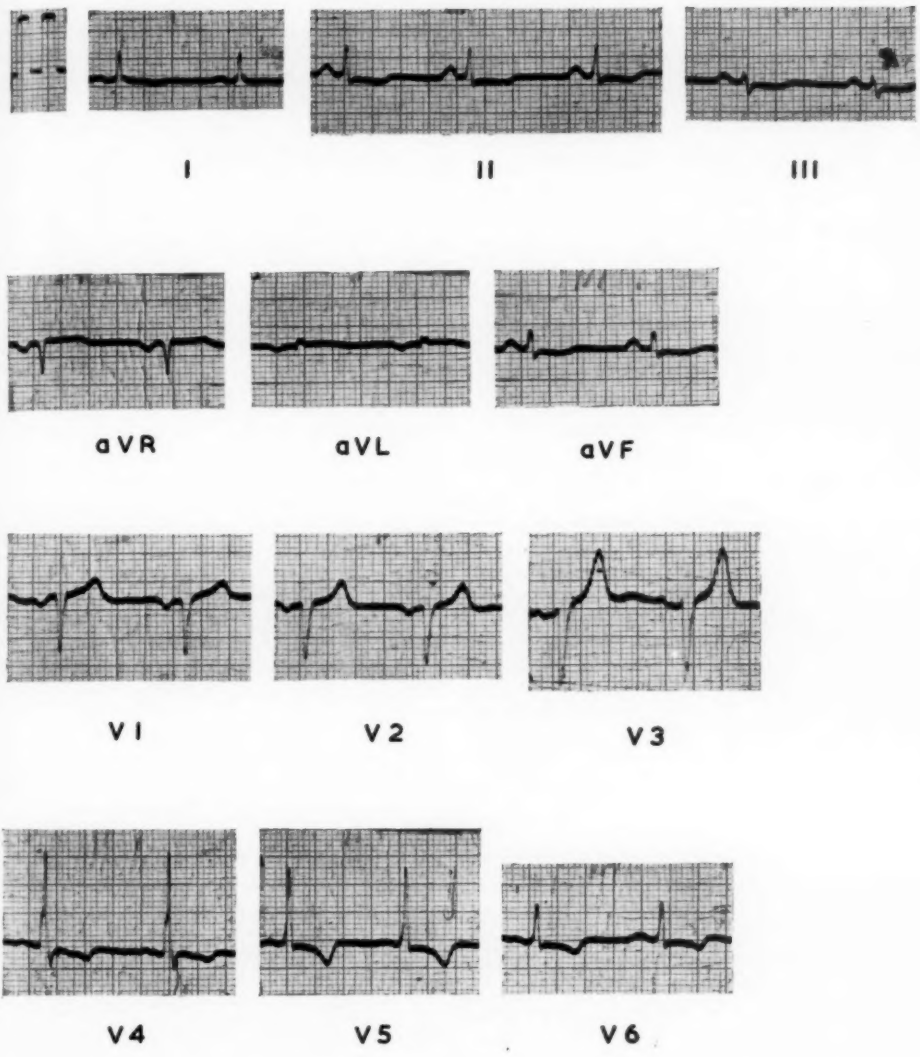

FIG. 5 (*continued*).

CASE 209

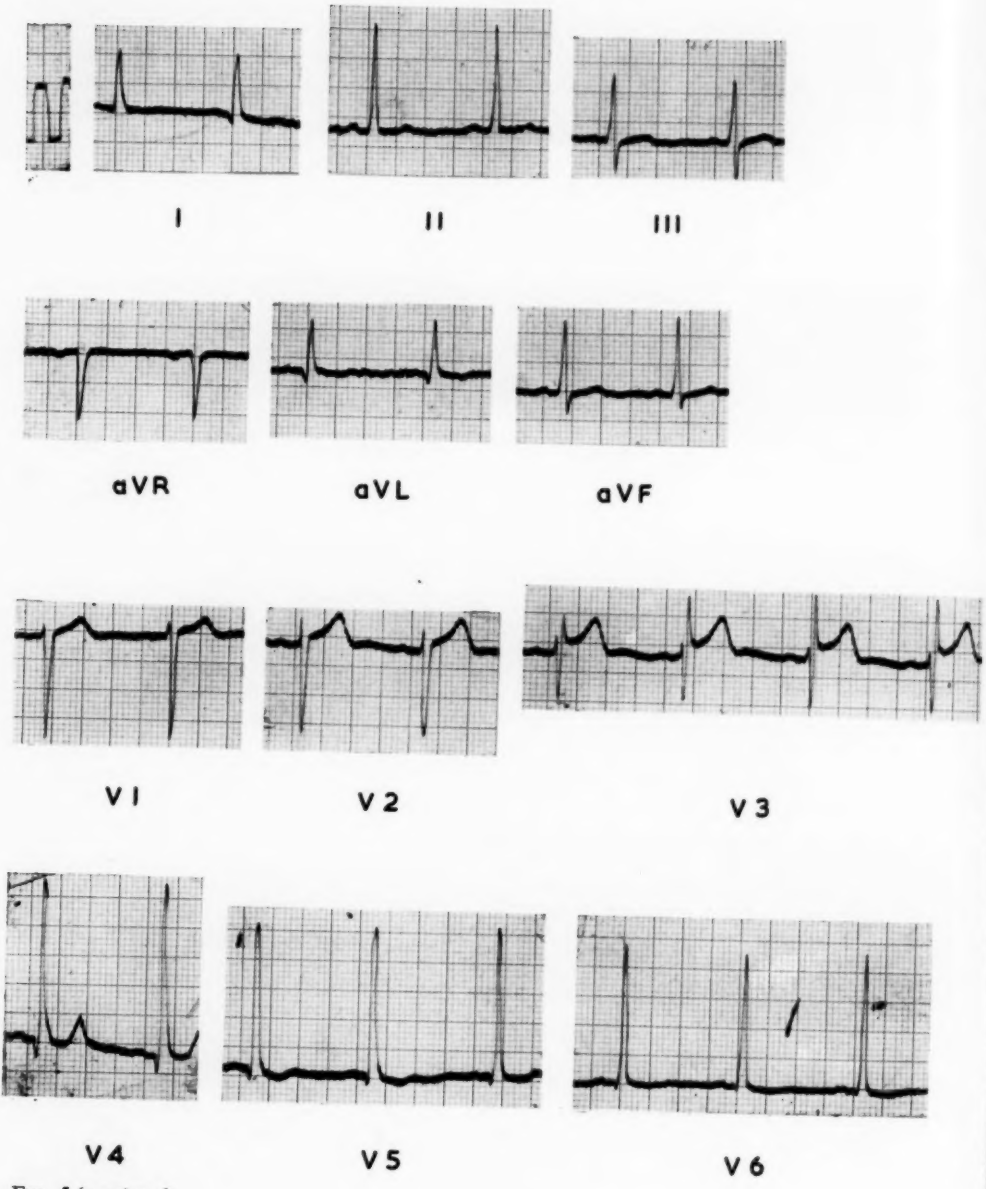

FIG. 5 (*continued*).

In the last three cases in Group III there was no unanimity about the group to which the tracing belonged; further details are given in Fig. 6 (overleaf, pp. 118–120) and Table IX. In all three Observer A, at one or other of his assessments, expressed doubt as to the nature of the lesion; one of these, Case 160, was the only one in the entire series in which the particular abnormality first diagnosed by Observer A_1 failed to meet with agreement at any of the subsequent readings. The observers did not all agree that the tracings in Table IX were in fact abnormal, and they also disagreed in diagnosing Left Ventricular Hypertrophy or Myocardial Ischaemia. In Case 170 the possibility of Myocardial Infarction was raised by three observers.

Frequency of the Various Diagnoses made by Each Assessor

A summary of the diagnoses made at each assessment is given in Table X. In this survey Atrial Fibrillation without any obvious cause has been considered to be a consequence of coronary heart disease (Acheson and Acheson, 1958), as were the other four diagnoses listed in Table X. Observer D estimated the frequency of coronary heart disease on this basis to be 25 per cent.; the estimate at the other four assessments ranged from 34 per cent (A_1) to 42 per cent. (C). The diagnosis of Left Ventricular Hypertrophy was made most frequently at four of the five assessments, the overall frequency ranging from 8 to 21 per cent.; Myocardial Ischaemia was the next most common diagnosis, the frequency varying from 2 to 9 per cent. It has been shown above that there was a good deal of difficulty in distinguishing between Left Ventricular Hypertrophy, Myocardial Ischaemia, and Myocardial Infarction. Therefore it is a matter of some interest that, if the occasions when these diagnoses were made are added, the figures for each observer are A_1 15, A_2 15, B 18, C 17, and D 10; there is less variation in the frequency of the diagnoses when they are combined than when they were taken separately.

TABLE X

GROUPING AND DIAGNOSIS OF 53 ECGs AT FIVE INDEPENDENT ASSESSMENTS

Assessment	A_1	A_2	B	C	D
Group I Normal	27	31	30	27	35
Group II ? Abnormal	8	2	2	4	5
Group III Abnormal	18	20	21	22	13
Fibrillation	3	3	3	3	3
Infarction	1	3	2	2	1
Bundle branch Block	0	2	0	2	0
Left Ventricular Hypertrophy	10	6	11	10	4
Myocardial Ischaemia	1	4	5	4	5
LVH or MI	3	2	0	0	0
Infarct or MI	0	0	0	1	0
Total	53	53	53	53	53

Discussion

The first matter which this investigation set out to examine was the observer error of Observer A on the first occasion he read the tracings. Before discussing his performance in detail it should be remembered that while Assessment A_1 was made from the entire tracing, all other assessments were made from two or three complexes selected from each lead; thus the fact that the readings were not being made from identical material may contribute to some extent to observer error and variation. This unfortunate difference was unavoidable because the complete tracings were not considered sufficiently

TABLE IX

INTERPRETATIONS OF THREE GROUP III ECGs ABOUT WHICH THERE WAS SECOND DEGREE DISAGREEMENT (SEE FIG. 6)

Case No.	Age (yrs)	Blood Pressure		Interpretations of Observers				
		Systolic	Diastolic	A_1	A_2	B	C	D
160	73	160	85	Left Ventricular Hypertrophy *or* Myocardial Ischaemia	Incomplete Right Bundle Branch Block	Normal	Incomplete Right Bundle Branch Block	Normal
170	72	195	95	Left Ventricular Hypertrophy *or* Myocardial Ischaemia ? Infarct	Old Anterior Infarct	Left Ventricular Hypertrophy ? Infarct	Left Ventricular Hypertrophy	Normal
172	76	140	90	Left Ventricular Hypertrophy	? Ventricular Hypertrophy *or* Myocardial Ischaemia ? Infarct	? Myocardial Ischaemia	Normal	? Myocardial Ischaemia

CASE 160

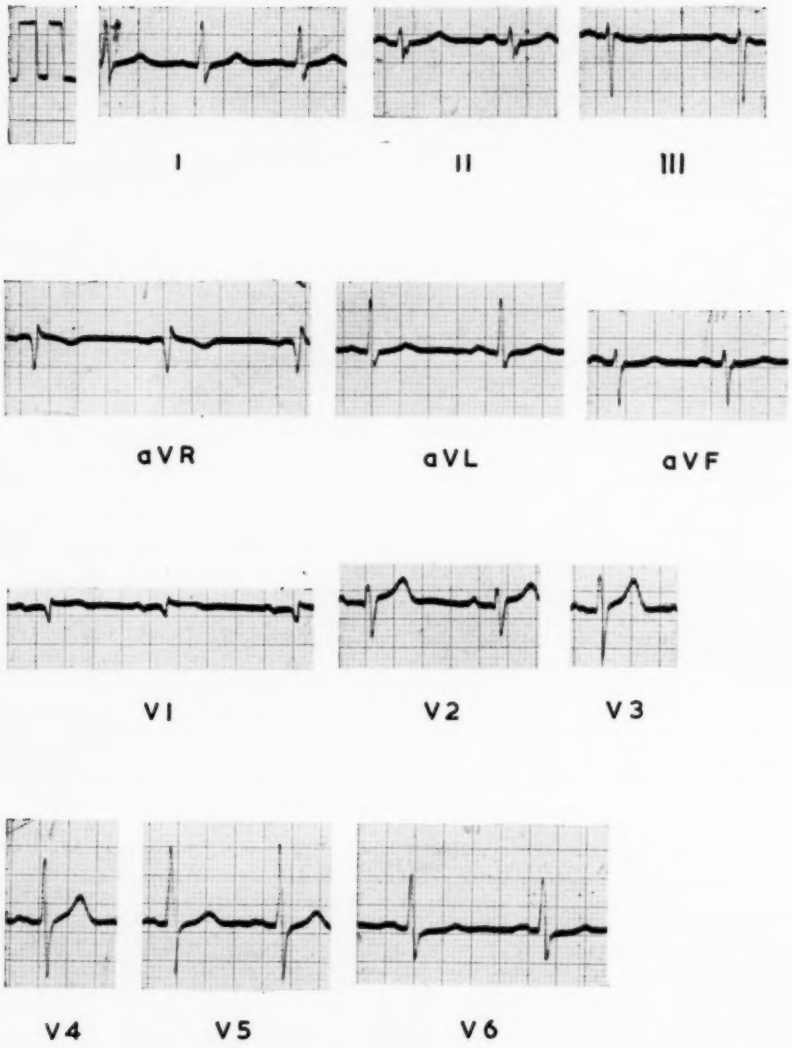

FIG. 6.—Three ECGs in Group III about which there was second degree disagreement in grouping (see also Table IX).

CASE 170

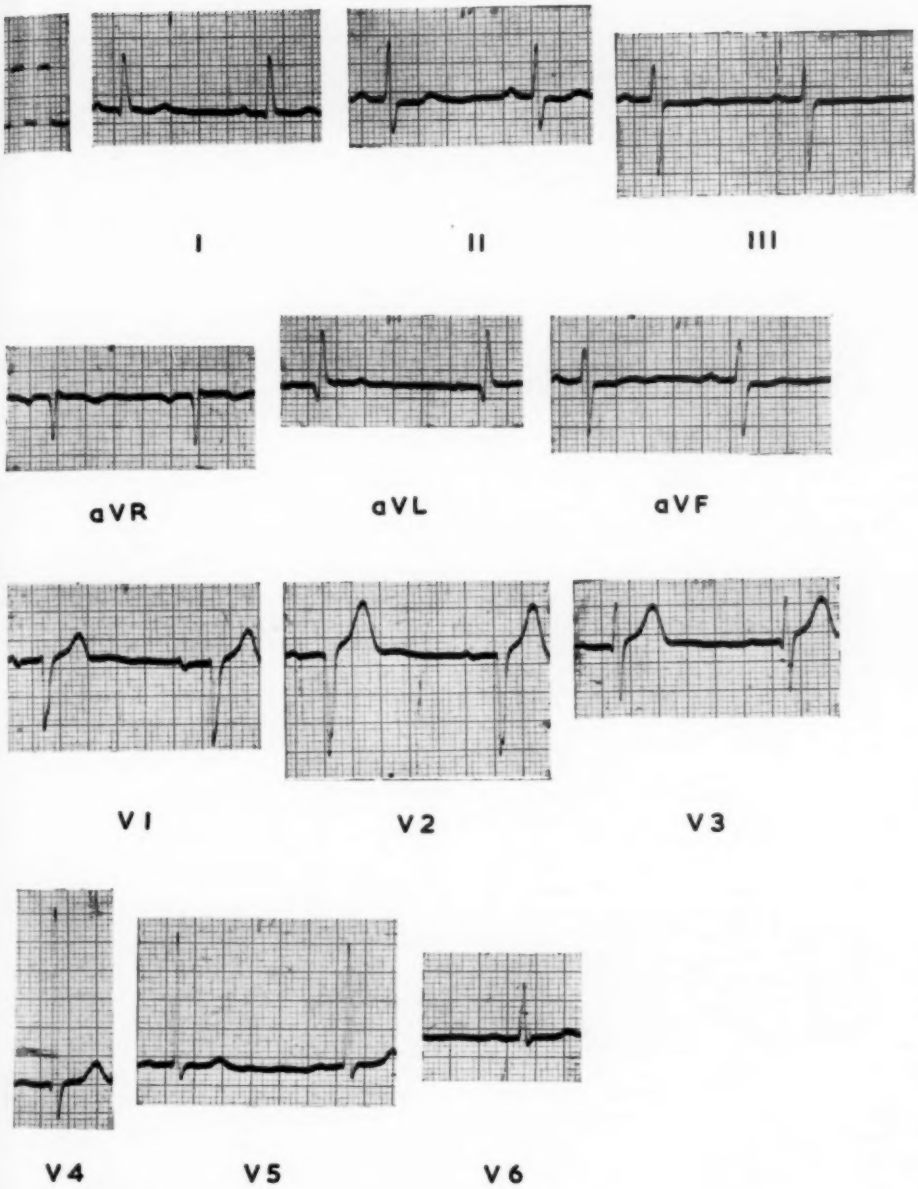

FIG. 6 (*continued*).

CASE 172

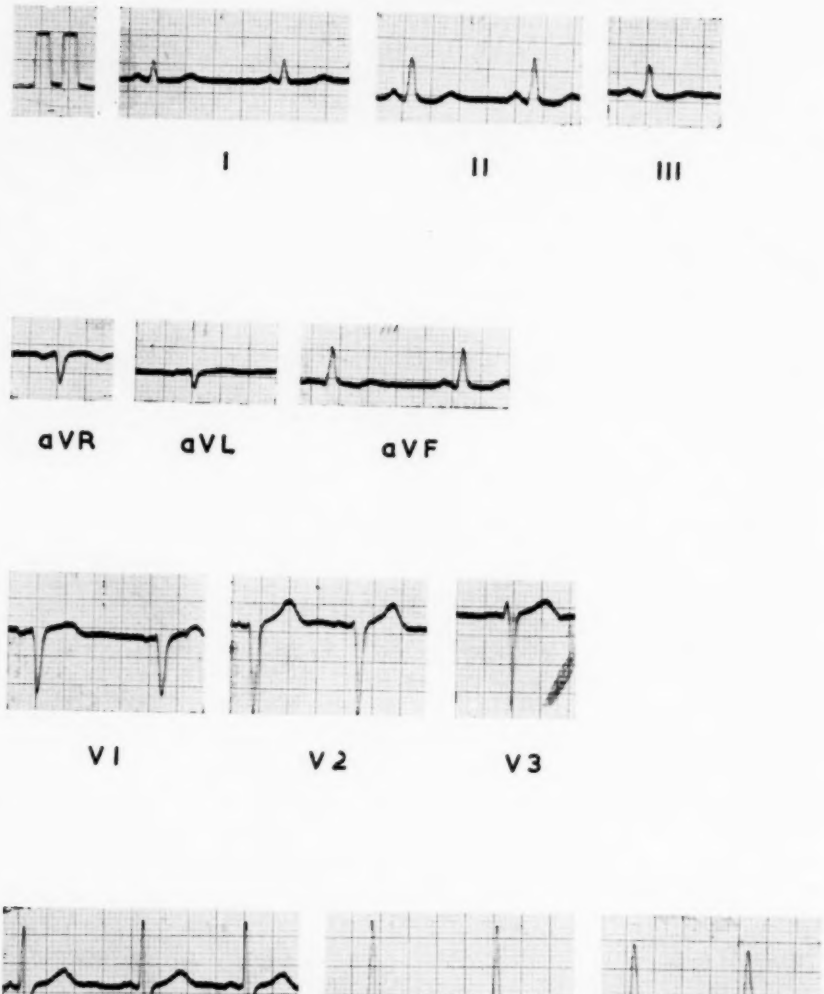

FIG. 6 (*continued*).

robust to withstand the handling and many postal journeys which were necessary for the study. On the other hand it was not feasible—nor from the point of view of the survey as a whole desirable—to mount more than 220 ECGs, many of which included lengthy "after exercise" tracings. Two to three complexes are usually considered to be adequate when resting ECGs are being used in the epidemiology of coronary heart disease (W.H.O., 1959) but, as Table IV suggests, the effect of mounting the ECGs seems to have been to increase the number of second degree disagreements (and therefore the observer error) and to reduce the first degree disagreements (and therefore the observer variation). It seemed to make Observer A more positive in his classification of certain "difficult" ECGs, which, to judge from the difference of opinion they evoked among the other observers, did not warrant such positivity.

It has been indicated in the text that the ability of Observer A can be measured in various ways. Table IV shows that, as far as grouping is concerned, he agreed with himself on 83 per cent. of the tracings, and also that the other observers agreed with his assessment of the mounted tracings in 84 per cent. of their assessments. It is shown, too, that if the question of whether or not the ECGs were mounted is set aside, and Assessment A_1 is compared with all the rest, second degree disagreement arose on only seven out of 212 occasions, that is to say, on this criterion his error was 3 per cent.

The suggestion that the error of an individual observer might under any circumstances be as low as 3 per cent. would at first sight appear to be in contradiction to the conclusion drawn by Davies (1958), in a similar study, that no arbitrary line can be drawn between normal and abnormal tracings. This is not, however, the case for several reasons; first, Davies had nine skilled observers each of whom assessed each ECG twice, giving a total of eighteen assessments of each tracing, and dissent is more likely to be found among eighteen than among five. Therefore the difference between a level of unanimity in 32 out of 53 (60 per cent.) in the present study and of 29 per cent. in his is not so great as it seems.

Secondly, the assessors in the present study were told the blood pressure of the subject, whereas Davies (1958) gave no clinical information whatsoever. This item of information was included in this investigation because it is one of the few objective clinical procedures and so is unlikely to introduce any element of false bias. Since the present data were collected, Bronte-Stewart and Pickering (1959) and W.H.O. (1959) have stated that they consider the revelation of blood pressure reasonable when ECGs are being interpreted in studies such as this.

Finally, Davies insisted that each tracing should be placed emphatically in one of three categories of "normal", "Infarction", and "All Abnormalities Other than Infarction", whereas the observers in the present study were asked to arrive at a diagnosis, paying special attention to the presence or absence of coronary heart disease. When they were given their instructions they were not offered the opportunity of classing a tracing as "? abnormal", but they did, in fact, use such a classification to a greater or lesser extent, and no subsequent attempt was made to induce them to be more definite. Presumably, however, as has already been argued, if they had been coerced into placing the ECGs into two categories only, the observer error would have been increased, although the variation might have been reduced. This difference between the present analysis and that of Davies (1958) stemmed from the different intentions behind the two studies; whereas Davies set out to establish whether "unlike the chest film, electrocardiography is a test free from observer variation", the present purpose was primarily to examine the performance of an individual assessor.

Bearing these differences in mind, there can be no doubt that the present findings offer full support to the conclusion of Davies that "it is an illusion to believe there can be any arbitrary line between normal and abnormal tracings, or between abnormal and infarction tracings".

The sources of variation and error are more difficult to determine, and the most positive statement that can be made on the present evidence is that Atrial Fibrillation seems to bring about clearly distinguishable changes in the ECG because this diagnosis was offered by all the observers in three cases, but was never offered by any of them at any other time. Infarction was diagnosed definitely nine times and on a tenth occasion Observer C could not decide whether the pattern of the tracing was due to Infarction or Ischaemia; five of these diagnoses were associated with a single tracing, giving an agreement level of 50 per cent. It also seems that there is a pattern which is acceptable as signifying Left Ventricular Hypertrophy, because there was unanimity about this diagnosis in four tracings—yet apart from these twenty occasions on which all the observers diagnosed Left Ventricular Hypertrophy there were a further 21 occasions when it was diagnosed without universal agreement (agreement level 49 per cent.). In contrast, there was agreement about Myocardial Ischaemia in only five of the

nineteen occasions (26 per cent.) on which it was definitely diagnosed; in the cases on which the observers disagreed whether the tracing was or was not Left Ventricular Hypertrophy, however, Myocardial Ischaemia was nearly always the second most popular diagnosis and *vice versa*. Thomas, Cochrane, and Higgins (1958) have also drawn attention to the difficulty experienced by independent observers in differentiating between these two diagnoses. Third on the list in this border-line group was Myocardial Infarction; W.H.O. (1959) suggested that "probable" Myocardial Infarction can be distinguished from "possible" Myocardial Infarction on the basis of changes in the Q-wave, but it was to the assessment of just these changes that Davies (1958) attributed most observer variation*. For these reasons, in the absence of aortic valvular lesions or some other contraindication, there would seem to be a strong argument in epidemiological studies of old men for grouping all cases where the tracing suggests Infarction, Left Ventricular Hypertrophy, or Myocardial Ischaemia together, and labelling them "Coronary Heart Disease", because in any age group coronary heart disease and hypertension are often found together. There is not the need in surveys for the precise diagnosis and prognosis which form the basis of sound clinical practice, and on the present evidence an attempt to subdivide the population whose ECGs show evidence of coronary or ischaemic lesions into smaller groups, will lead to spurious accuracy.

Finally, it appears that not only is there a continuum between the normal and the abnormal ECG, but there seem also to be continua between all the diagnoses offered by the assessors (except Fibrillation) and between each of these and normality (Fibrillation again excepted). Thus the case of Fletcher and Oldham (1959) and of Davies (1958) is strengthened in pressing for the recognition of the importance of observer variation by epidemiologists and clinicians alike.

Summary

56 electrocardiograms from a population sample of old men were chosen at random from a series of over 220; three were rejected as incomplete or imperfect, leaving a total of 53 for study. These ECGs were mounted on cards and assessed independently once by each of four observers, one of whom, Observer A, had already interpreted the tracings before mounting and had also read all the other ECGs in the series. All four observers were clinical cardiologists, cardiological epidemiologists, or both, and they made a total of 265 assessments. The only clinical data they were given were age, blood pressure, and whether or not digitalis was being taken.

The principal assessor, Observer A, upheld his own opinion whether a tracing was "normal", "? abnormal", or "abnormal" in 83 per cent. if his second assessments. Similarly, his assessments of the mounted ECGs were upheld by the three other observers in 84 per cent. of 159 assessments. His own second assessment, and the assessments of the other three observers set the grouping error of his first assessment at 3 per cent.

The only diagnosis made with unanimity throughout was Atrial Fibrillation, about which there was 100 per cent. agreement in three cases, and which was not otherwise diagnosed. The complete agreement level for Myocardial Infarction was five out of the ten occasions on which the diagnosis was made (50 per cent.), for Left Ventricular Hypertrophy, twenty out of 41 (49 per cent.), and for Myocardial Ischaemia five out of nineteen (26 per cent.).

There were therefore many tracings which were unanimously agreed to be abnormal, but variously diagnosed as Left Ventricular Hypertrophy, Myocardial Ischaemia, or Myocardial Infarction.

Thus it may be concluded that, unless clinical examination reveals some lesion such as aortic valvular disease, which requires that they should be diagnosed otherwise, there is a case, in epidemiological studies of old men in whom two or three of these conditions may occur together, for grouping all such tracings as "Coronary Heart Disease".

My thanks are due to Dr. Donald Acheson, U.S. Veterans Administration, Washington; Dr. Ian Higgins, M.R.C. Pneumoconiosis Research Unit, Llandough, Glamorganshire; Dr. Grant Lee, Radcliffe Infirmary, Oxford, and Dr. A. J. Thomas, United Cardiff Hospitals, for their kindness in assessing the electrocardiograms. I am also most grateful to the directors and medical staff of Messrs. Arthur Guinness Son and Co. Ltd., Dublin, for making the survey possible, and to Prof. W. J. E. Jessop for his advice and encouragement. Prof. W. S. Walton kindly read the text.

* See footnote † to p. 99.

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CLIMATE, AIR POLLUTION, AND MORTALITY

BY

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Seasonal variation in disease has attracted the interest and speculation of writers since the days of Hippocrates (Greenwood, 1921). More recently Farr, in the course of his wide-ranging researches on 19th century vital statistics, published several studies relating to medical meteorology and drew attention to the excessive mortality that often occurred during periods of very low temperature (Farr, 1885). During the 20th century other workers have demonstrated the close nature of this association between temperature and mortality, and the London fog disaster of 1952 (Logan, 1953) focused attention on the effects of air pollution and the role it may play in any general relationship between climate and mortality.

The present studies were planned to make some further investigation of associations in time between mortality, particularly respiratory mortality, and different indices of meteorological conditions and atmospheric pollution. They were based on data relating to both a metropolitan environment, London Administrative County, and a rural environment in East Anglia. The East Anglia data covered all rural districts of Norfolk, Suffolk, Cambridge, and the Isle of Ely, and were included to provide a control study in which possible effects of gross urban pollution would be eliminated from associations between meteorological conditions and mortality. In both areas, the various associations were measured by a series of time correlations relating weekly mortality to temperature, absolute humidity, and visibility, while for the metropolitan area it was also possible to examine correlations between mortality and both sulphur dioxide and smoke pollution. Relevant data (Appendix) were supplied from official sources:

(*a*) Weekly mortality figures by age and cause from the General Registrar Office;

(*b*) Daily meteorological readings by the Meteorological Office of the Air Ministry, Harrow;

(*c*) Daily pollution measurements by the Fuel Research Station (D.S.I.R.).

The choice of a time unit for the study, one week, was governed by this being the shortest period for which data on deaths were readily available. Thus weekly deaths, by cause and age, provided indices of mortality, and basic data on each of the other variables were grouped to form parallel series of weekly indices.

The indices derived from the London data were as follows:

(1) *Mortality.*—Weekly deaths from (*a*) bronchitis and pneumonia and (*b*) heart disease.

(2) *Temperature.*—Weekly average of mean daily minimum temperature (°F.) derived from recordings at ten stations in London A.C.

(3) *Absolute Humidity.*—Weekly average mean daily vapour pressure recordings (m.b.) at Kew.

(4) *Sulphur Dioxide.*—Weekly average of mean daily SO_2 concentrations (p.p.m.) derived from recordings at five stations in London A.C.

(5) *Smoke Pollution.*—Weekly average of mean daily smoke recordings (mg/m³) at five stations in London A.C.

(6) *Fog.*—Weighted average* of number of hours per week with visibility less than 440 yards, from recordings at Kingsway and Croydon.

* For each week the number of hours of visibility less than 440 yards were weighted as follows:

Number of hours of visibility less than 44 yards	..	$\times 10 = a$
Number of hours of visibility 44–219 yards ..	..	$\times 2 = b$
Number of hours of visibility 220–439 yards	..	$\times 1 = c$
		$a+b+c$

The value $(a+b+c)$ was derived separately for Kingsway and Croydon data, and the average of these two values was taken weekly index of visibility.

For the rural districts of East Anglia, comparable indices were derived, in like manner, for mortality, temperature, humidity, and fog, the basic data for the last three indices coming from recording stations in the rural area.

The period of study extended over the years 1947 to 1954 but, since any relationship between mortality and the factors under investigation seemed likely to be more evident during the winter, the analysis was restricted to the winter months of those years. Each "winter" was arbitrarily defined as the 26-week period centred in the end of one calendar year and the beginning of the next, and, using this definition, data from seven winters, 1947–48 to 1953–54 inclusive, were available for analysis.

All correlations were calculated separately for each winter and the overall pattern of association between variables was obtained by averaging correlations over the seven winters. An alternative approach would have been to average the indices for each of the 26 winter weeks and to use the composite, or averaged, data to measure correlation between the different variables. This alternative approach was however rejected since, with the known broad seasonal influences on each of the factors concerned, it would inevitably have caused an artificial smoothing of time trends for all variables and artificially high correlations between them. The method adopted, *i.e.* averaging correlations rather than indices over the seven winters, also seemed to make more allowance for possible changes in mortality patterns over the period, and for any factors which might have an effect in the interplay between variables in some winters but not in others. As will be noted later, the investigation itself provided some evidence of such a factor, namely influenza, which when prevalent appeared to distort some of the associations under study.

Throughout the main part of the analysis, weekly deaths were correlated with indices representing climatic and pollution factors in the same week and also with similar indices of conditions in each of the six preceding weeks.

Results

Primary Correlations—London Administrative County

Temperature.—Respiratory mortality appeared to be most closely associated with temperature conditions of the 2 weeks immediately preceding death (Table I).

Table I

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AND RELATED TEMPERATURE INDICES

Temperature Relating to	Weekly Deaths from Bronchitis and Pneumonia		
	Under 1 yr	45 yrs and Over	65 yrs and Over
Same Week	−0·220	−0·499	−0·491
Week −1	−0·459	−0·720	−0·706
Week −2	−0·404	−0·719	−0·710
Week −3	−0·320	−0·668	−0·655
Week −4	−0·322	−0·640	−0·625
Week −5	−0·305	−0·628	−0·614
Week −6	−0·330	−0·580	−0·570

When weekly deaths from bronchitis and pneumonia, at 45 years and over, were correlated with temperature, a correlation around −0·5 was obtained with temperature index of the same week perature of the preceding week. This level was maintained using temperature 2 weeks before death and was followed by a consistent decline when deaths were related to temperature of successively earlier weeks. While consistent the decline was a gradual one and the correlation coefficient between mortality and temperature conditions 6 weeks before remained considerable. An exactly similar pattern emerged from the analysis relating to deaths at 65 years and over. At the other end of the age scale (under one year) association between deaths and temperature was less marked, the highest coefficient being under −0·5. Although correlation levels were lower in this youngest age group, the same general pattern remained evident and the highest coefficient were again attained with temperature of Weeks −1 and −2.

When correlations for individual winters were examined separately (Table II, opposite), some divergences from the general trend became apparent. Alternate winters (1947–48, 1949–50, 1951–52, 1953–1954) displayed the pattern already described, but trends in the remaining winters (1948–49, 1950–51, 1952–53) did not show the same consistency. A common factor to each of the winters in the first group was their relatively low influenza mortality compared with winters in the second group (Table III, opposite). These data were used to separate winters according to concurrent influenza mortality and the average temperature correlations for "low" and "high" influenza winters were examined separately (Table IV, opposite).

For low influenza winters the pattern displayed was similar to, but more accentuated than, that

TABLE II

LONDON ADMINISTRATIVE COUNTY: CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES IN THE WINTERS 1947–48 TO 1953–54 INCLUSIVE

Temperature Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	−0·361	−0·540	−0·631	−0·277	−0·583	−0·411	−0·688
Week −1	−0·726	−0·736	−0·836	−0·486	−0·835	−0·530	−0·890
Week −2	−0·807	−0·571	−0·806	−0·570	−0·786	−0·649	−0·842
Week −3	−0·670	−0·582	−0·716	−0·640	−0·746	−0·652	−0·667
Week −4	−0·568	−0·606	−0·623	−0·754	−0·662	−0·632	−0·632
Week −5	−0·676	−0·709	−0·579	−0·795	−0·577	−0·438	−0·621
Week −6	−0·780	−0·659	−0·494	−0·781	−0·464	−0·319	−0·561

TABLE III

RECORDED DEATHS FROM INFLUENZA, AT 45 YRS AND OVER, IN THE WINTERS 1947–48 TO 1953–54

Winter	Number of Deaths from Influenza	
	London Administrative County	Rural Districts of East Anglia
1947–48	60	14
1948–49	251	60
1949–50	171	27
1950–51	717	284
1951–52	90	9
1952–53	481	70
1953–54	54	7

obtained in the overall analysis. The association between mortality and temperature of 1 or 2 weeks previously, as measured by a coefficient of −0·8, was closer, and there was a sharper decline in size of correlation when mortality was related to temperature of earlier weeks. In contrast, the average temperature correlations from high influenza winters did not reveal the same regular pattern. They no longer reached their maximum with temperature of the 2 weeks immediately preceding death and, while there was a gradual increase in correlation back to that with temperature 4 weeks before death, they did not display any clear cut trend comparable to that for the low influenza winters. A possible explanation of the distorting effect of influenza suggested by this broad difference is that, when prevalent, the rise and the fall of the disease may have caused sufficient fluctuation of concurrent mortality, particularly from respiratory causes, to disturb the normal relationship between temperature and mortality. In any event it is clear that this "influenza effect" has been partly responsible for the artificially high average correlations obtained in the overall analysis between mortality and temperature conditions relating to 5 or 6 weeks before death. It is probable, however, that even for low influenza winters these correlations remain elevated by a close inter-association between temperatures of successive weeks.

TABLE IV

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES FOR "LOW" AND "HIGH" INFLUENZA WINTERS

Temperature Relating to	"Low" Influenza Winters	"High" Influenza Winters
Same Week	−0·567	−0·409
Week −1	−0·822	−0·584
Week −2	−0·810	−0·597
Week −3	−0·700	−0·625
Week −4	−0·621	−0·664
Week −5	−0·613	−0·647
Week −6	−0·575	−0·586

In addition to correlation based on deaths from bronchitis and pneumonia, parallel series were calculated correlating temperature with weekly mortality from bronchitis and heart disease respectively (Table V). For both mortality groupings, the pattern of correlations was essentially similar to that presented by deaths from bronchitis and pneumonia. Their time trend was, however, a little sharper than for the bronchitis and pneumonia correlations averaged over the seven winters. This was more noticeable in the correlations based on cardiac deaths and may have been due to the trends of mortality from heart disease being less affected by the presence or absence of influenza.

TABLE V

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND HEART DISEASE, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES

Temperature Relating to	Weekly Deaths at 45 yrs and Over	
	Bronchitis	Heart Disease
Same Week	−0·538	−0·552
Week −1	−0·727	−0·744
Week −2	−0·694	−0·688
Week −3	−0·638	−0·553
Week −4	−0·620	−0·559
Week −5	−0·604	−0·544
Week −6	−0·559	−0·519

The separate analysis of heart disease data for high and low influenza winters (Table VI) provided some evidence supporting this explanation in that even in winters with the higher prevalence of influenza peak correlations were still attained with temperatures of the two weeks immediately preceding death.

TABLE VI

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM HEART DISEASE, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES FOR "LOW" AND "HIGH" INFLUENZA WINTERS

Temperature Relating to	"Low" Influenza Winters	"High" Influenza Winters
Same Week	−0·611	−0·473
Week −1	−0·798	−0·672
Week −2	−0·708	−0·661
Week −3	−0·549	−0·559
Week −4	−0·508	−0·627
Week −5	−0·481	−0·627
Week −6	−0·478	−0·574

Humidity.—Correlations between weekly mortality and absolute humidity (Table VII) were so closely similar to the temperature associations that it is unnecessary to present any further detailed description of trends displayed. For all practical purposes, both as regards pattern of correlations and levels attained, the humidity correlations mirrored those of temperature. They rose to a peak coefficient of −0·7 with indices relating to conditions of the 2 weeks preceding death, the association was less strong for deaths under one year, and there was little variation between the different mortality groups examined. Separate analysis of winters with high and low influenza mortalities again demonstrated the difference between them and suggested that, as for temperature, the humidity correlation pattern was disturbed during periods when influenza was prevalent. This distorting effect was again more marked on correlations based on respiratory mortality.

TABLE VII

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AND RELATED HUMIDITY INDICES

Humidity Relating to	Weekly Deaths from Bronchitis and Pneumonia		
	Under 1 yr	45 yrs and Over	65 yrs and Over
Same Week	−0·213	−0·508	−0·485
Week −1	−0·451	−0·722	−0·705
Week −2	−0·404	−0·727	−0·718
Week −3	−0·329	−0·674	−0·665
Week −4	−0·332	−0·633	−0·617
Week −5	−0·299	−0·636	−0·621
Week −6	−0·341	−0·583	−0·573

Sulphur Dioxide.—The average sulphur dioxide correlations were smaller and presented a less definite pattern than the temperature or humidity series. In general, the coefficient obtained was of the order of +0·5 and varied little when weekly mortality from bronchitis and pneumonia was correlated with sulphur dioxide concentrations of different preceding weeks (Table VIII).

TABLE VIII

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AND RELATED SULPHUR DIOXIDE POLLUTION INDICES

SO_2 Pollution Relating to	Weekly Deaths from Bronchitis and Pneumonia		
	Under 1 yr	45 yrs and Over	65 yrs and Over
Same Week	+0·297	+0·465	+0·444
Week −1	+0·382	+0·498	+0·501
Week −2	+0·351	+0·469	+0·464
Week −3	+0·342	+0·473	+0·454
Week −4	+0·345	+0·559	+0·546
Week −5	+0·372	+0·531	+0·521
Week −6	+0·309	+0·511	+0·496

Thus, while the highest correlation was achieved with indices relating to 4 weeks before death, the peak in time was ill-defined, and this lack of any real time trend among sulphur dioxide correlations contrasted strongly with the regular pattern of the temperature and humidity series. There was no material difference between correlations relating to mortality at 45 years and over and those relating to deaths at older ages, and, while associations with mortality under one year again appeared to be weaker than for the older age groups, this difference was less marked than in the temperature and humidity series. Separate analyses relating sulphur dioxide pollution to bronchitis mortality and mortality from heart disease yielded results differing little from those based on deaths from bronchitis and pneumonia (Table IX).

TABLE IX

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND HEART DISEASE, AT 45 YRS AND OVER, AND RELATED SULPHUR DIOXIDE POLLUTION INDICES

SO_2 Pollution Relating to	Weekly Deaths at 45 yrs and Over	
	Bronchitis	Heart Disease
Same Week	+0·481	+0·435
Week −1	+0·474	+0·441
Week −2	+0·451	+0·442
Week −3	+0·451	+0·378
Week −4	+0·548	+0·490
Week −5	+0·512	+0·450
Week −6	+0·499	+0·469

Correlation with deaths from heart disease was, however, slightly lower than with respiratory mortality.

TABLE X

LONDON ADMINISTRATIVE COUNTY: CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED SULPHUR DIOXIDE POLLUTION INDICES FOR WINTERS 1947–48 TO 1953–54 INCLUSIVE

SO_2 Pollution Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	+0·530	+0·307	+0·618	+0·440	+0·348	+0·578	+0·431
Week −1	+0·666	+0·430	+0·675	+0·386	+0·443	+0·580	+0·303
Week −2	+0·426	+0·440	+0·640	+0·456	+0·605	+0·362	+0·354
Week −3	+0·426	+0·412	+0·562	+0·546	+0·659	+0·264	+0·445
Week −4	+0·717	+0·582	+0·468	+0·610	+0·578	+0·378	+0·579
Week −5	+0·785	+0·582	+0·399	+0·612	+0·535	+0·305	+0·496
Week −6	+0·693	+0·486	+0·278	+0·691	+0·478	+0·335	+0·616

Examination of sulphur dioxide correlations in separate winters revealed some high individual values but failed to demonstrate any consistent pattern (Table X). The trends showed considerable variation from year to year and the suggestion in the overall analysis of a minor peak at Week −4 was not consistently present in individual winters. Nor did it appear that influenza exerted any strong effect on the pattern of association, since average correlations for low and high influenza winters (Table XI) were similar to one another and to those obtained in the overall analysis.

TABLE XI

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED SULPHUR DIOXIDE POLLUTION INDICES FOR "LOW" AND "HIGH" INFLUENZA WINTERS

SO_2 Pollution Relating to	"Low" Influenza Winters	"High" Influenza Winters
Same Week	+0·482	+0·442
Week −1	+0·522	+0·465
Week −2	+0·506	+0·419
Week −3	+0·523	+0·407
Week −4	+0·586	+0·523
Week −5	+0·554	+0·500
Week −6	+0·516	+0·504

Smoke Pollution.—None of the average smoke correlations (Table XII) exceeded a value of +0·4. They resembled sulphur dioxide correlations in that apart from a more definite increase between correlations relating to the same and immediately preceding weeks they display no regular trend over the time scale studied. No major differences were apparent between series for different age and mortality groupings.

TABLE XII

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AND RELATED SMOKE POLLUTION INDICES

Smoke Pollution Relating to	Weekly Deaths from Bronchitis and Pneumonia		
	Under 1 yr.	45 yrs and Over	65 yrs and Over
Same Week	+0·112	+0·196	+0·186
Week −1	+0·334	+0·358	+0·348
Week −2	+0·282	+0·352	+0·355
Week −3	+0·319	+0·347	+0·341
Week −4	+0·331	+0·391	+0·394
Week −5	+0·338	+0·385	+0·381
Week −6	+0·357	+0·386	+0·377

Though some of the individual winters showed trends similar to temperature correlations, other winters provided very different results and there was no evidence of a pattern common to all (Table XIII).

TABLE XIII

LONDON ADMINISTRATIVE COUNTY: CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED SMOKE POLLUTION INDICES FOR WINTERS 1947–48 TO 1953–54 INCLUSIVE

Smoke Pollution Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	+0·049	+0·320	+0·254	+0·332	+0·129	+0·462	−0·175
Week −1	+0·232	+0·557	+0·522	+0·354	+0·321	+0·603	−0·114
Week −2	+0·169	+0·539	+0·549	+0·436	+0·300	+0·415	+0·059
Week −3	+0·179	+0·486	+0·514	+0·472	+0·339	+0·344	+0·092
Week −4	+0·413	+0·420	+0·469	+0·599	+0·209	+0·440	+0·187
Week −5	+0·484	+0·432	+0·409	+0·713	+0·095	+0·354	+0·210
Week −6	+0·457	+0·301	+0·306	+0·739	+0·080	+0·339	+0·481

Visibility.—The correlation studies did not reveal any strong association between weekly mortality and related fog indices. The average correlations (Table XIV) were small, both absolutely and relative to those attained between mortality and other measures of pollution and climate. None of the average fog correlations reached a level as high as +0·2 and no definite time trend was noted in any of the mortality groupings examined.

TABLE XIV

LONDON ADMINISTRATIVE COUNTY: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AND RELATED FOG INDICES

Fog Relating to	Weekly Deaths from Bronchitis and Pneumonia		
	Under 1 yr	45 yrs and Over	65 yrs and Over
Same Week	+0·029	+0·054	+0·047
Week −1	+0·142	+0·132	+0·144
Week −2	+0·188	+0·060	+0·083
Week −3	+0·101	+0·006	+0·013
Week −4	+0·128	+0·104	+0·108
Week −5	+0·151	+0·138	+0·133
Week −6	+0·127	+0·151	+0·134

Correlations relating to separate winters (Table XV) were equally inconsistent and through one winter produced individual coefficients as high as +0·4, the data from another showed consistently negative correlations between mortality and visibility. Repetition of the analysis using a second index of visibility, an *unweighted* instead of a *weighted* average of number of hours of fog (visibility <440 yards), again failed to demonstrate any general association between mortality and visibility. The results were equally inconsistent and largely followed the pattern, or lack of pattern, described above.

PRIMARY CORRELATIONS—RURAL DISTRICTS OF EAST ANGLIA

Because of the difference in size of the two populations, the numbers of deaths occurring in the rural area were less than in London and several of the mortality groupings used in the London analysis provided very few weekly deaths. It was not therefore possible to use as wide a range of mortality groupings by cause and age and in East Anglia deaths from heart disease formed the main group providing adequate numbers. Deaths classified in this category amounted, on average over the seven winters, to about sixty per week at age 45 years and above, and this group was used as the principal mortality index for correlation studies in the rural area. Correlations with deaths from bronchitis and pneumonia were also calculated, though the numbers available, an average of eleven to twelve per week, were rather small for any adequate demonstration of relevant associations.

Temperature.—The association between temperature and weekly mortality from heart disease presented a not dissimilar pattern to that obtained from the London data and was again closest when mortality was related to the temperature of the immediately preceding weeks (Table XVI).

TABLE XVI

RURAL DISTRICTS OF EAST ANGLIA: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS PNEUMONIA AND HEART DISEASE, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES

Temperature Relating to	Weekly Deaths at 45 yrs and Over	
	Bronchitis and Pneumonia	Heart Disease
Same Week	−0·530	−0·616
Week −1	−0·634	−0·698
Week −2	−0·658	−0·635
Week −3	−0·647	−0·616
Week −4	−0·641	−0·619
Week −5	−0·675	−0·614
Week −6	−0·663	−0·631

A further parallel was the gradual decrease in average correlations with temperature of successively earlier weeks. The range of variation was however somewhat less than for London. Since rural respiratory deaths were small in number, it was not surprising that their correlation with temperature did

TABLE XV

LONDON ADMINISTRATIVE COUNTY: CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED FOG INDICES FOR WINTERS 1947–48 TO 1953–54 INCLUSIVE

Fog Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	+0·018	+0·150	+0·039	+0·128	−0·213	+0·436	−0·177
Week −1	+0·261	+0·099	+0·365	+0·032	−0·085	+0·422	−0·171
Week −2	−0·038	+0·048	+0·187	−0·034	−0·009	+0·150	+0·117
Week −3	−0·117	−0·071	−0·028	+0·044	−0·017	+0·139	+0·093
Week −4	+0·039	+0·055	+0·105	+0·092	−0·119	+0·437	+0·118
Week −5	+0·203	+0·078	+0·199	+0·319	−0·268	+0·313	+0·123
Week −6	+0·141	+0·092	+0·130	+0·478	−0·265	+0·261	+0·217

TABLE XVII

RURAL DISTRICTS OF EAST ANGLIA: CORRELATIONS BETWEEN WEEKLY DEATHS FROM HEART DISEASE, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES FOR WINTERS 1947–48 TO 1953–54 INCLUSIVE

Temperature Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	−0·537	−0·553	−0·572	−0·548	−0·635	−0·699	−0·766
Week −1	−0·708	−0·753	−0·708	−0·528	−0·810	−0·653	−0·723
Week −2	−0·607	−0·621	−0·726	−0·590	−0·727	−0·548	−0·628
Week −3	−0·399	−0·724	−0·707	−0·656	−0·651	−0·684	−0·489
Week −4	−0·355	−0·585	−0·677	−0·766	−0·622	−0·704	−0·626
Week −5	−0·533	−0·599	−0·574	−0·790	−0·468	−0·776	−0·558
Week −6	−0·600	−0·727	−0·585	−0·759	−0·523	−0·715	−0·450

not conform to the same regular pattern. Separate examination of the data for individual winters (Table XVII) and in relation to concurrent levels of influenza mortality suggested that the influenza effect already noted in the London area might also have been a factor in the disturbance of rural associations. During low influenza winters the average correlations between respiratory deaths and temperature (Table XVIII) reverted to a pattern which, having regard to the small number of deaths involved, was not inconsistent with other temperature series.

TABLE XVIII

RURAL DISTRICTS OF EAST ANGLIA: AVERAGE CORRELATIONS BETWEEN WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA, AT 45 YRS AND OVER, AND RELATED TEMPERATURE INDICES FOR "LOW" AND "HIGH" INFLUENZA WINTERS

Temperature Relating to	"Low" Influenza Winters	"High" Influenza Winters
Same Week	−0·541	−0·515
Week −1	−0·673	−0·583
Week −2	−0·693	−0·610
Week −3	−0·657	−0·634
Week −4	−0·594	−0·703
Week −5	−0·602	−0·773
Week −6	−0·587	−0·765

Humidity.—Attention has been drawn to the close parallel between humidity and temperature correlations derived from the London data. This close relationship was also evident in the East Anglia analysis, where humidity correlations (Table XIX) broadly followed the patterns of temperature correlations with respect to coefficient values, time trends, and variations between individual winters.

Visibility.—The negative findings of the metropolitan visibility analysis were repeated in the rural correlations, which again failed to demonstrate any general association between weekly mortality and the fog index employed in the study. Correlations were small, their signs varied, and the same irregular variation was apparent between and within series for separate winters.

Thus, from both metropolitan and rural analysis, it appeared that weekly mortality was most closely associated with temperature and humidity of the immediately preceding weeks. The close agreement between the two analyses suggested that urban pollution, present in London but absent in East Anglia, did not have any marked effect on the general association between temperature or humidity, and mortality.

PARTIAL CORRELATIONS

In order further to assess inter-relationships between the various factors under study, a limited number of partial first order correlations were calculated using the indices most closely associated with mortality in the primary analysis. These indices were those of temperature and humidity relating to the week immediately preceding death, and sulphur dioxide and smoke pollution indices 4 weeks before

TABLE XIX

RURAL DISTRICTS OF EAST ANGLIA: CORRELATIONS BETWEEN WEEKLY DEATHS FROM HEART DISEASE, AT 45 YRS AND OVER, AND RELATED HUMIDITY INDICES FOR WINTERS 1947–48 TO 1953–54 INCLUSIVE

Humidity Relating to	Winters						
	1947–48	1948–49	1949–50	1950–51	1951–52	1952–53	1953–54
Same Week	−0·534	−0·525	−0·555	−0·562	−0·580	−0·617	−0·712
Week −1	−0·712	−0·744	−0·726	−0·561	−0·801	−0·628	−0·680
Week −2	−0·590	−0·603	−0·724	−0·602	−0·723	−0·582	−0·635
Week −3	−0·372	−0·715	−0·693	−0·627	−0·649	−0·663	−0·490
Week −4	−0·416	−0·642	−0·695	−0·721	−0·630	−0·722	−0·594
Week −5	−0·626	−0·614	−0·644	−0·776	−0·535	−0·739	−0·589
Week −6	−0·676	−0·670	−0·628	−0·717	−0·599	−0·724	−0·538

death. The mortality experience incorporated in this analysis was that from respiratory and heart disease at 45 years and over, and, as with the primary correlations, partial correlations were calculated for each winter and then averaged over the seven winters of the study period (Tables XX and XXI).

TABLE XX

LONDON ADMINISTRATIVE COUNTY: AVERAGE FIRST ORDER PARTIAL CORRELATIONS BETWEEN SPECIFIED FACTORS AND WEEKLY DEATHS FROM BRONCHITIS AND PNEUMONIA AT 45 YRS AND OVER

Correlation With	Total Coefficient	First Order Partial Coefficient with Given Factors held Constant			
		Temperature	Humidity	SO_2 Pollution	Smoke Pollution
Temperature	−0·720	—	−0·150	−0·607	−0·652
Humidity ..	−0·722	−0·188	—	−0·573	−0·643
SO_2 Pollution ..	+0·559	+0·366	+0·318	—	+0·388
Smoke Pollution ..	+0·391	+0·245	+0·184	+0·109	—

TABLE XXI

LONDON ADMINISTRATIVE COUNTY: AVERAGE FIRST ORDER PARTIAL CORRELATIONS BETWEEN SPECIFIED FACTORS AND WEEKLY DEATHS FROM HEART DISEASE AT 45 YRS AND OVER

Correlation With	Total Coefficient	First Order Partial Coefficient with Given Factors held Constant			
		Temperature	Humidity	SO_2 Pollution	Smoke Pollution
Temperature	−0·744	—	−0·149	−0·662	−0·692
Humidity ..	−0·754	−0·190	—	−0·646	−0·695
SO_2 Pollution ..	+0·490	+0·199	+0·140	—	+0·314
Smoke Pollution ..	+0·330	+0·178	+0·097	+0·010	—

Temperature and Humidity.—The two indices appeared to be almost completely dependent on one another. This was reflected in the very high correlation between them (+0·953) and the reduction in coefficient when either was correlated with mortality with the other held constant For both respiratory deaths and those from heart disease primary correlations of the order −0·7 (−0·8 in the four low influenza winters) were reduced to −0·2 or less. This was true whether humidity was held constant in the correlation between mortality and temperature or *vice versa* and. although not shown in the Tables, it was equally evident in partial correlations based on the East Anglia data. Removal of pollution factors, sulphur dioxide or smoke, did not cause any marked fall in either temperature or humidity correlations. For respiratory mortality, primary coefficients were reduced from −0·7 to −0·6, and the reduction in size of coefficient was even less for mortality from heart disease. The effect of separating sulphur dioxide from temperature and humidity correlations was slightly more pronounced than that of removing smoke from these correlations.

Sulphur Dioxide and Smoke.—The interdependence between these two indices, as measured by a correlation coefficient of +0·689, was less marked than between temperature and humidity. The relevant partial correlations suggested that mortality was more closely associated with sulphur dioxide than with smoke pollution indices. Thus, for respiratory mortality, the smoke correlation was reduced to +0·1 when sulphur dioxide was held constant, while the sulphur dioxide correlation when smoke was held constant yielded a coefficient of almost +0·4. The same difference was evident from partial correlations based on deaths from heart disease. For both mortality groupings, the separation of temperature or humidity from smoke correlations reduced the latter to +0·2 or less. The same reduction was apparent in sulphur dioxide correlations relating to deaths from heart disease, but those between sulphur dioxide and respiratory mortality still remained between +0·3 and +0·4 after removing temperature and humidity from the correlation.

In general, this limited partial correlation analysis confirmed the indices of temperature and humidity as those showing the closest association with mortality from respiratory and heart disease. For both factors, the respective coefficients were little reduced by partialling out either of the pollution indices. On the other hand it was clear that the nearly complete association between temperature and humidity prevented any discrimination as to their relative importance. Of the pollution indices, sulphur dioxide appeared to be the more important, in that the partial correlations with respiratory mortality, though small compared with those of temperature and humidity, remained appreciable.

Relationship between Mortality and Pollution Indices at Different Temperature Levels

The close association between mortality and temperature was again apparent when average weekly deaths in London were analysed according to temperature of the week preceding death (Table XXII). For each of the causes of death examined, mortality increased with decreasing temperature.

although there were differences in the relative increase for different causes. The largest occurred amongst deaths from bronchitis which, on average, increased three-fold over the range of temperature used for this analysis. Over the same temperature range deaths from pneumonia were doubled, and those from heart disease rose by about 50 per cent. While deaths in the rural districts were smaller in number, their pattern of variation with temperature was broadly similar to that shown by the London data.

TABLE XXII

AVERAGE WEEKLY DEATHS FROM CARDIO-RESPIRATORY DISEASE, AT 45 YRS AND OVER, RELATED TO TEMPERATURE INDEX OF PRECEDING WEEK

Area	Temperature of Preceding Week (°F.)	Average Weekly Deaths			
		Bronchitis	Pneumonia	Heart Disease	Respiratory + Heart Disease
London Administrative County	Less than 32°	161	62	258	481
	32°–	140	63	227	430
	36°–	93	46	198	337
	40° and Over	51	31	162	244
Rural Districts of East Anglia	Less than 32°	10	9	74	93
	32°–	8	6	65	79
	36°–	8	6	62	76
	40° and Over	4	2	45	51

The visibility data available for both metropolitan and rural areas allowed an extension of this temperature analysis and an assessment of relative effects of fog in the two areas. In both, weekly mortality within temperature groupings was subdivided according to the number of hours of related fog (visibility < 440 yards). The main feature of this analysis for the East Anglia area (Table XXIII) was the lack of any definite association between visibility and mortality under constant conditions of temperature. It appeared that, within each of the different ranges of temperature, mortality levels were unaffected by variation in the number of hours of related fog. The one possible exception occurred for bronchitis mortality in weeks with a related temperature index below 32°F. At this temperature, average deaths from bronchitis rose from eight for weeks with no preceding fog to fourteen for weeks preceded by at least 20 hours of fog.

The pattern emerging from the parallel analysis of London data contrasted sharply with that for East Anglia (Figure, overleaf) and suggested a definite association between fog and mortality when concurrent temperatures were very low (Table XXIV). When related temperature was below 32°F. the weeks with the highest fog rating showed an average bronchitis mortality three times greater than that for weeks with the lowest fog rating. This relationship between fog and mortality was also present, though in lesser degree, for deaths from pneumonia and heart disease, the former being more than doubled and the latter increased by about one third over the same fog range. In the next temperature grouping (32–35°F.), a mortality gradient remained evident for bronchitis and possibly pneumonia, but with higher temperatures (above 36°F.), the apparent association between fog and mortality from both respiratory and heart disease had disappeared. A corollary of the mortality gradients within the lower temperature groupings was a notable accentuation of the bronchitis mortality gradient with temperature among weeks with the highest fog rating. Under these fog conditions, average weekly deaths from bronchitis increased from fifty with related temperature above 40°F. to over 300 with temperature below 32°F.

TABLE XXIII

RURAL DISTRICTS OF EAST ANGLIA: AVERAGE WEEKLY DEATHS FROM CARDIO-RESPIRATORY DISEASE, AT 45 YRS AND OVER, RELATED TO TEMPERATURE AND FOG INDICES OF PRECEDING WEEK

Preceding Week		Average Weekly Deaths			
Temperature (°F.)	Hours of Fog	Bronchitis	Pneumonia	Heart Disease	Respiratory + Heart Disease
<32°	0	8	9	79	96
	1–19	10	7	71	88
	20+	14	11	74	99
32°–	0	8	5	67	80
	1–19	8	6	64	78
	20+	8	6	63	77
36°–	0	8	6	59	73
	1–19	8	6	64	78
	20+	7	5	61	73
40°+	0	4	3	45	52
	1–19	4	2	46	52
	20+	3	4	43	50

TABLE XXIV

LONDON ADMINISTRATIVE COUNTY: AVERAGE WEEKLY DEATHS FROM CARDIO-RESPIRATORY DISEASE, AT 45 YRS AND OVER, RELATED TO TEMPERATURE AND FOG INDICES OF PRECEDING WEEK

Preceding Week		Average Weekly Deaths			
Temperature (°F.)	Hours of Fog	Bronchitis	Pneumonia	Heart Disease	Respiratory + Heart Disease
<32°	0	100	40	249	389
	1–19	102	50	228	379
	20+	309	93	329	730
32°–	0	106	51	213	370
	1–19	139	65	241	445
	20+	161	68	218	447
36°–	0	112	53	210	375
	1–19	83	43	191	317
	20+	90	45	209	345
40°+	0	54	32	166	251
	1–19	48	28	159	236
	20+	50	37	155	241

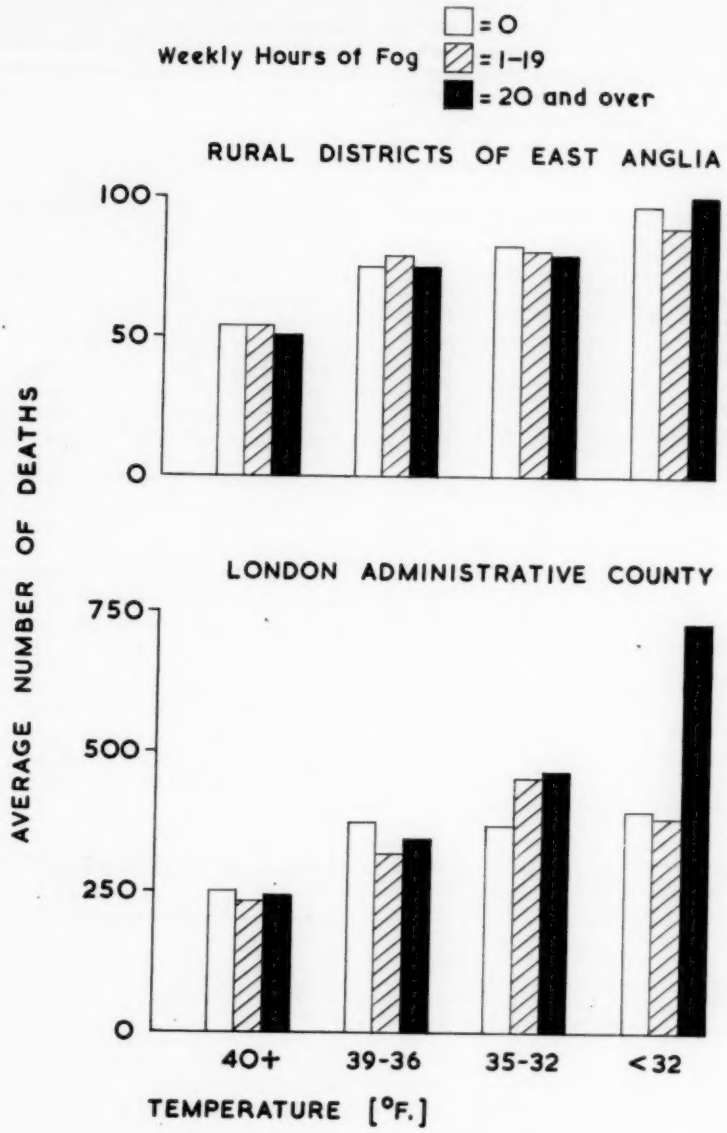

FIGURE.—Mortality related to temperature and weekly hours of fog in London and East Anglia.

Comparable analyses were carried out relating mortality to the other pollution indices available for the London area. Although the different arbitrary gradings prevent detailed comparisons between the three pollution indices, the general patterns emerging from the sulphur dioxide and smoke analyses were similar to that obtained from the visibility study. The data relating to all three indices suggested that the closest association between mortality and pollution was found when temperatures were very low. There were however some differences between trends displayed by the separate analyses. From the sulphur dioxide data (Table XXV), it appeared that association with mortality was largely confined to deaths from respiratory causes, and that within each temperature range average weekly deaths from heart disease varied little with differing levels of related sulphur dioxide pollution. Moreover, with respect to respiratory mortality the association, while again most marked for bronchitis and under low temperature conditions, did not as in the visibility analysis completely disappear in the upper ranges of temperature scale.

TABLE XXV

LONDON ADMINISTRATIVE COUNTY: AVERAGE WEEKLY DEATHS FROM CARDIO-RESPIRATORY DISEASE, AT 45 YRS AND OVER, RELATED TO TEMPERATURE AND SULPHUR DIOXIDE POLLUTION INDICES OF PRECEDING WEEK

Preceding Week		Average Weekly Deaths			
Temperature (°F.)	SO_2 Pollution (p.p.m.)	Bronchitis	Pneumonia	Heart Disease	Respiratory + Heart Disease
<32°	<0·100	142	67	281	490
	0·100–	99	45	231	375
	0·150+	236	80	287	603
32°–	<0·100	89	45	222	356
	0·100–	134	63	228	425
	0·150+	236	70	227	533
36°–	<0·100	81	41	186	308
	0·100–	91	44	199	334
	0·150+	106	55	207	368
40°+	<0·100	38	23	152	213
	0·100–	61	36	170	267
	0·150+	60	38	169	267

The parallel smoke analysis (Table XXVI) did not provide any new features. As with sulphur dioxide no close association with mortality from heart disease was apparent and although there was again evidence of an excess respiratory mortality with increasing pollution, this was only present in the smoke data at the lower end of the temperature scale.

TABLE XXVI

LONDON ADMINISTRATIVE COUNTY: AVERAGE WEEKLY DEATHS FROM CARDIO-RESPIRATORY DISEASE, AT 45 YRS AND OVER, RELATED TO TEMPERATURE AND SMOKE POLLUTION INDICES OF PRECEDING WEEK

Preceding Week		Average Weekly Deaths			
Temperature (°F.)	Smoke Pollution (mg./m.³)	Bronchitis	Pneumonia	Heart Disease	Respiratory + Heart Disease
<32°	<0·250	121	54	265	440
	0·250–	98	54	225	377
	0·400+	217	69	280	566
32°–	<0·250	145	72	239	456
	0·250–	121	57	223	401
	0·400+	165	71	228	464
36°–	<0·250	103	52	209	364
	0·250–	90	44	193	327
	0·400+	85	43	198	326
40°+	<0·250	41	25	155	221
	0·250–	63	36	169	268
	0·400+	48	34	166	248

DISCUSSION

The associations between mortality trends and rise and fall of seasonal temperature have been analysed in several investigations based on London mortality experience and weather records. Young (1924) studied deaths from respiratory disease among children under 5 years over the period 1976–1919. Correlations with different climatic factors were, in this study, calculated separately for each of the winter weeks, *i.e.* for all 44 "first" weeks, for 44 "second" weeks, and seriatim for the successive 26 weeks of the arbitrarily defined winter. From the results he concluded that, of the factors examined which included rainfall and relative humidity, temperature appeared "to exercise the greatest influence on predisposing to an increased mortality from . . . bronchitis". Mortality was found to be more closely related to the temperature conditions of the week preceding death than to the conditions of the same week or of 2 weeks before. Russell (1926), using long-term mortality (1870–1890) from respiratory disease at 55 years and over, and Woods (1927) working with monthly groupings of penumonia mortalities at age 40 and above for the years 1855–1904, employed similar methods to demonstrate the clear inverse relationship between number of weekly (or monthly) deaths and related temperatures. The different approach of Wright and Wright (1945) measured temperature mortality association by deriving *average* mortality rates from bronchitis and pneumonia and related *average* temperature indices for each of the 26 winters weeks over the years 1923–1929. Correlations based on these indices and relating mortality to the temperature of the same and preceding weeks

were strikingly high and demonstrated a peak association between mortality and temperature 2 weeks (for mortality under 2 years) or 4 weeks (for mortality over 55 years) before death.

The close agreement between the general findings of these studies of mortality-temperature relationships did not however extend to their assessments of the *degree* of association as measured by levels of coefficient attained; *e.g.* where Young found an average correlation of $-0{\cdot}3$ between mortality and the temperature of the preceding week, a corresponding coefficient of almost $-0{\cdot}9$ was demonstrated by Wright and Wright. It seems possible that such discrepancies were largely the result of differences in method, particularly in relation to the grouping of data before correlation. Thus the numerically relatively low coefficients obtained by Young may have been partly due to the distorting effect of trends in mortality from bronchitis and pneumonia over the long period studied, while the use of averaged data may have artificially elevated the correlations of Wright and Wright. The method employed in the current investigation, calculation of all correlations separately for individual winters, was adopted in an attempt to minimize such effects and also the possible distortion of association by any seasonal factor, *e.g.* influenza, prevalent in some winters but not in others.

The limitations inherent in all correlation studies are well recognized, and the hazards of inferring causation from association are perhaps particularly treacherous in a field where all the variables are themselves so closely linked together. Within these limitations, however, the current study has clearly indicated that, among the climatic and pollution indices examined, those relating to temperature and absolute humidity were most closely associated with mortality. Findings with respect to temperature were consistent with those already established but suggested that the association was equally evident for mortality from respiratory and heart disease, and in both rural and metropolitan environments. Highest correlations (up to $-0{\cdot}8$) were achieved with indices relating to the 2 weeks immediately preceding death and correlation with conditions of successively earlier weeks declined from this peak. A similar time trend of temperature correlations relating to the weeks before death was noted by Wright and Wright. In the present study this was accentuated and there was also some internal evidence against its being rejected as an unexplained artefact in that the regular pattern of association was blurred or even lost in winters when influenza was prevalent.

In contrast to the many investigations measuring relationships with temperature, associations between seasonal variations in mortality and absolute humidity have been largely neglected. In another context, Waddy (1952), speculating on the different weather conditions associated with seasonal peaks of respiratory infection in tropical and temperate countries, put forward evidence that the common climatic factor linking these peaks was low absolute humidity. It was suggested that in temperate winters the seasonal low absolute humidity might exert a drying action on the mucosa of the upper respiratory tract and so lower its vitality and resistence to infection. The present results have found weekly mortality to be associated equally closely with absolute humidity and temperature, but the very high correlation between these two variables prevented any discrimination as to their relative importance.

Among the pollution factors examined, sulphur dioxide demonstrated the highest correlations and appeared to be more closely associated with respiratory mortality than with deaths from heart disease. Average correlations between weekly mortality and all pollution factors were, however, invariably lower than those for temperature and humidity and did not display the same regular pattern. Further, the mortality trends in relation to differing degrees of pollution within separate temperature ranges strongly suggested that the more serious effects of metropolitan fog, as measured by excessive increases in mortality, occurred only when fog was accompanied by very low temperatures. This finding is consistent with the conclusions of Russell (1924) and also allows some reconciliation between the results of the present analysis and the more dominant role of pollution suggested by short-term studies of individual fog episodes.

I am indebted to the General Register Office for supplying the East Anglian Mortality data, and to Dr. E. T. Wilkins of the Fuel Research Station (D.S.I.R.) and Mr. R. G. Veryard of the Air Ministry Meteorological Office, Harrow, for their co-operation in allowing the use of records from these sources. My thanks are also due to Mrs. Ann Martin who did most of the abstracting of records and computing.

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APPENDIX

METROPOLITAN AND RURAL DATA ON WHICH INDICES INCORPORATED IN CORRELATION STUDIES WERE BASED

LONDON ADMINISTRATIVE COUNTY

(*a*) *Mortality.*—Weekly deaths by age from the following causes:

Before 1950	1950 Onwards
Bronchitis (21)	Bronchitis (B 32)
Pneumonia (22)	Pneumonia (B 31)
Influenza (9)	Influenza (B 30)
Coronary Disease and Angina Pectoris (19·3)	Coronary and Arteriosclerotic Heart Disease (B 26a)
Myocardial Degeneration (19·2)	Other Myocardial Degeneration (B 26c)

Figures in brackets after each specified cause relate to the abbreviated lists of the International Statistical Classifications of Diseases, Injuries, and Causes of Death (1938 and 1948)—as adapted and used by the Registrar General.

(*b*) *Temperature.*—Daily minimum temperature (°F.) recorded at the following ten stations:

Kew	East Ham	Regents Park
Wealdstone	Camden Square	Hampstead
St. James Park	Kensington	Greenwich
	Croydon	

(*c*) *Humidity.*—Daily mean vapour pressure (m.b.) recordings at Kew.

(*d*) *Visibility.*—Number of hours daily when visibility was (*i*) less than 44 yards, (*ii*) 44–219 yards, (*iii*) 220–440 yards, from recordings at Kingsway and Croydon.

(*e*) *Sulphur Dioxide.*—Daily SO_2 (p.p.m) readings from five recording stations:

Erith	Becton	County Hall
Greenwich	Carteret	St.-Grosvenor Rd.

Carteret Street and Grosvenor Road are adjacent stations in Westminster and together provided a continuous record over the seven winters.

(*f*) *Smoke Pollution.*—Daily concentrations of smoke (mg./m.^{3}) from the same recording stations as those recording sulphur dioxide.

COMBINED RURAL DISTRICTS OF NORFOLK, SUFFOLK, CAMBRIDGE, AND ISLE OF ELY

(*a*) *Mortality.*—Weekly deaths by age from the following causes:

Cause of Death	Office Code of the Registrar General	
	Before 1950	1950 Onwards
Bronchitis	521–549	500–502
Pneumonia	551–556	490–493
Influenza	060–063	480–483
Heart and Circulatory Diseases	400–508	410–468

(*b*) *Temperature.*—Daily minimum temperatures (°F.) from recording stations at Felixstowe, Mildenhall, and West Raynham.

(*c*) *Humidity.*—Daily mean vapour pressure (m.b.) recordings at Felixstowe, Mildenhall, and West Raynham.

(*d*) *Visibility.*—Number of hours daily when visibility was (*i*) less than 44 yards, (*ii*) 44–219 yards, (*iii*) 220–440 yards, from recordings at Mildenhall and West Raynham.

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ANENCEPHALY IN UNIOVULAR TWINS

REPORT OF A CASE

BY

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Anencephaly is a relatively common congenital malformation occurring with a frequency of up to 0·3 per cent. of births. Malpas (1937) reported an incidence of 0·308 per cent. in 13,964 births, and Record and McKeown (1949) from 158,307 births found an incidence of 0·23 per cent. Hurwitz (1955) reviewed the whole literature and included his own series of 5,264 births, among which he found fifteen cases of anencephaly, representing an incidence of 0·28 per cent.

Of greater interest than the occurrence merely of a particular type of malformation is its occurrence in one of a pair of uniovular twins. So far as the causes of malformations are concerned, they may be either hereditary or environmental, or a combination of both. When a malformation has occurred in one of a pair of uniovular twins it has been suggested that the causative factors must have been associated with the environment of the affected foetus, because both foetuses would have been malformed if an hereditary factor alone had been operating. This is not to imply that the genetic constitution of the twins is in no way responsible, but that without an additional environmental factor any genetic factor will not operate. That anencephaly can be caused by environmental factors is also suggested by experimental work (*e.g.* Ingalls, Curley, and Prindle, 1952; Hicks 1952). From the practical standpoint this means that certain malformations may be classed as preventable diseases, although it is difficult to see how satisfactory prophylactic measures can be taken at a time when the state of pregnancy cannot definitely be ascertained.

A case in which one of a pair of twins was anencephalic was reported by Josephson and Waller (1933). Here, however, both of the foetuses were abnormal; twin A was an iniencephalic and twin B was anencephalic. Moreover, there was no specific report on the placental membranes; "there was only one placenta, the babies were both females, and probably identical twins". These authors quoted a report by Thompson (1925) of a case of anencephaly in one of a pair of twins. In fact, in Thompson's case, the normal living child was a male and the one malformed foetus was not a true anencephalic but, rather, an acardiac acephalic. There was no comment on the state of the placental membranes.

Potter (1952) reported having seen two instances of twins in a single amniotic sac and attached to a single placenta in which one twin was normal while the other grossly abnormal. In one of these pairs the malformed foetus was anencephalic. The presence in this case of one chorion and one amnion seems definite evidence of an origin from a single ovum. When a single chorionic membrane is associated with two amniotic sacs it is usual to consider the twins as being uniovular; but this is not invariably true, it seems, for Potter and Crunden (1941) reported an instance of a monochorionic placenta associated with twins of the opposite sex. However, where there is a single chorion and the twins are of the same sex, it is extremely probable that the twins are uniovular.

The present report describes such an example of uniovular twins, both of them male; one was anencephalic and the other without malformation of any kind.

Case Report

The mother was aged 37 years and had had only one previous pregnancy; this had resulted in the birth of an healthy female infant. During her present

pregnancy there was no history of trauma or disease and she had received no inoculation. Exposure of the abdomen to *x* rays had been limited to a simple diagnostic procedure one week before delivery at which time the presence of twins including an anencephalic was confirmed. Her blood group was A rhesus negative, but during the course of pregnancy no rhesus antibodies had been demonstrated. The father was also aged 37 years and was in good health. There was no history of deformity occurring in the sibs, parents, or grandparents of either mother or father.

During the present pregnancy, vomiting had occurred early, and by the 30th week pronounced hydramnios and oedema of the ankles had developed. The pregnancy was terminated at the 35th week by artificial rupture of the membranes, and resulted in the spontaneous delivery of male twins (Figs 1 and 2) neither of which survived. The placenta, weighing 675 g., was expelled complete and consisted of one chorion and two amnions; from the form of the placenta (Fig. 3, overleaf) and the fact that the twins were of the same sex it was concluded that the twins were uniovular.

FIG. 1.—Twin 1, normal.

FIG. 2.—Twin 2, anencephalic.

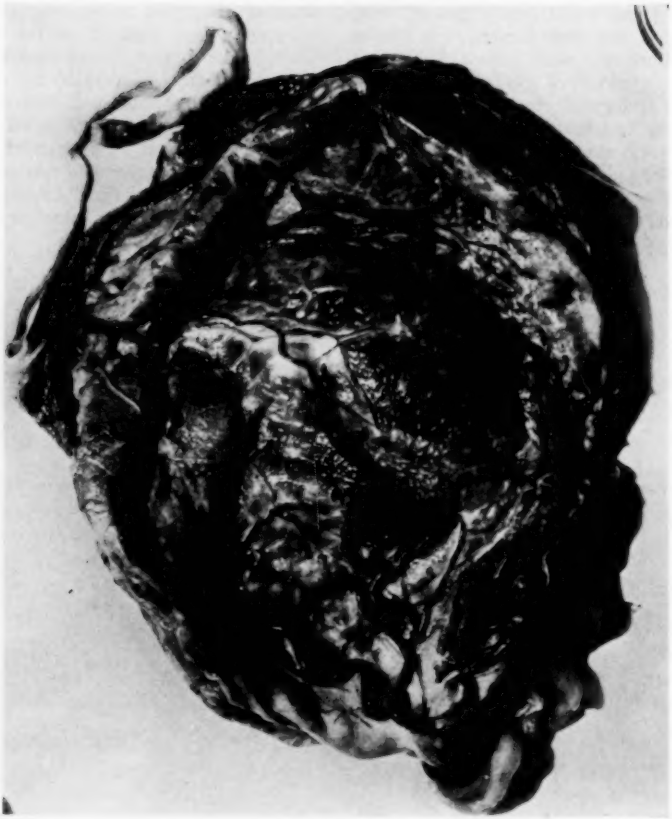

FIG. 3.—Placenta.

Post-mortem Findings:

Twin 1.—This male foetus weighed 526 g. and showed no obvious external or internal malformation. The weights of certain organs were, heart 4·1 g., liver 23·5 g., thymus 0·7 g., and paired adrenals 1·2 g. The lungs were unexpanded and airless.

Twin 2.—This foetus, also male, weighed 621 g. and had anencephaly with the facies characteristic of this condition. Over the lumbar spine was a tuft of hair and dissection revealed spina bifida occulta. No other malformation was found on external and internal examination. The heart weighed 8·6 g., liver 46·0 g., thymus 3·6 g., and paired adrenals 0·3 g. The lungs were unexpanded and airless, and histological examination failed to demonstrate in them any recognizable brain tissue.

SUMMARY

Anencephaly is described in one only of a pair of twins with a single chorion.

I am indebted to Mr. G. Davey of Shoreham for the photographs.

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MORTALITY FROM CEREBROVASCULAR ACCIDENTS AND HYPERTENSION IN THE REPUBLIC OF IRELAND

BY

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It is well known that hypertension may be associated with certain cerebrovascular accidents on the one hand, and with coronary heart disease on the other. The fact that two diseases tend to occur together in one patient, however, does not necessarily mean that they are similar in their epidemiology or mortality. A study of the mortality from coronary artery disease in the Republic of Ireland over the past 30 years has recently been published from this department (Acheson and Thornton, 1958), and it was felt that it might be instructive to undertake similar analyses of the vital statistics of the Republic for hypertension and cerebrovascular accidents and to compare them with those already published for coronary artery disease. Although it is recognized that mortality figures are a poor index of the prevalence of hypertension (W.H.O., 1959), they are probably a fair index of the incidence of cerebrovascular accidents taken as a group.

Data

The Irish data are again drawn from the annual reports of the registration of births, marriages, and deaths in the Republic of Ireland from 1926 to 1957, prepared on behalf of the Registrar General. The classification of cause of death is made in accordance with the International Statistical Classification, which has been changed three times since 1926. It proved impossible to obtain any continuity over the entire period in respect of hypertension, but some continuity has been obtainable for cerebrovascular accidents. The exact details of the list numbers adopted for the present study are presented in Table I.

TABLE I

DESCRIPTION OF DISEASES CLASSIFIED AS HYPERTENSION AND CEREBROVASCULAR ACCIDENTS

International Classification	Hypertension	Cerebrovascular Accidents
1926–1930 (Third Revision)		74a Cerebral Haemorrhage, Apoplexy 74b Cerebral Thrombosis and Embolism 75a Hemiplegia
1931–1939 (Fourth Revision)	102 Idiopathic Abnormalities of Blood Pressure	82a Cerebral Haemorrhage b Cerebral Thrombosis, Embolism c Cerebral Softening d Hemiplegia
1940–1949 (Fifth Revision)		83 Intracranial Lesions of Vascular Origin
1950–1956 (Sixth Revision)	440 Essential Benign Hypertension with Heart Disease 441 Essential Malignant Hypertension with Heart Disease 442 Hypertensive Heart Disease with Arteriolar Nephrosclerosis 443 Other and Unspecified Hypertensive Heart Disease 444 Essential Benign Hypertension without mentions of Heart 445 Essential Malignant Hypertension without mention of Heart 446 Hypertension with Arteriolar Nephrosclerosis without mention of Heart 447 Other Hypertensive Disease without mention of Heart	331 Cerebral Haemorrhage 332 Cerebral Embolus and Thrombosis

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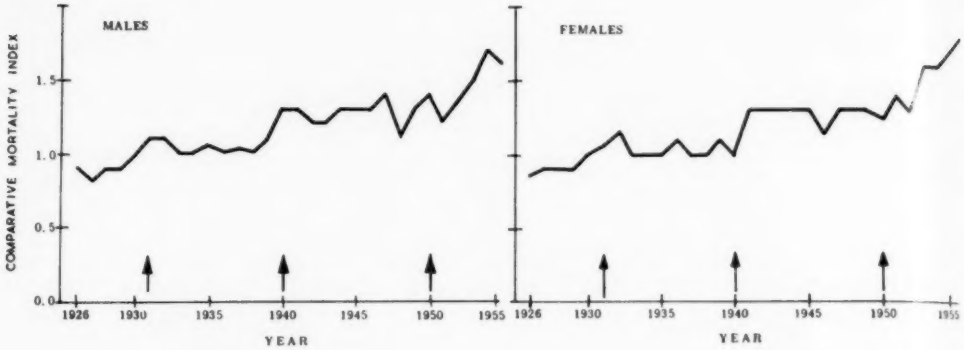

FIG. 1.—Comparative mortality indices for both sexes for cerebro-vascular accidents in the Republic of Ireland, 1926 to 1956.

It will be noted that the International List has never distinguished cerebral embolus and cerebral thrombosis, and that for the present purpose these two diagnoses have been grouped with cerebral haemorrhage.

SECULAR TREND

Comparative mortality indices (C.M.Is.) taking 1938 as unity, are shown for both sexes for cerebrovascular accidents in Fig. 1. There is a slight secular increase between 1926 and 1949, the index rising from 0·9 to 1·3 in both sexes. Between 1950 and 1956, however, the increase is much more rapid, rising from 1·3 to 1·8 in both sexes.

Changes in the International List have made it impossible to calculate the comparative mortality indices for hypertension. The age-specific death rates for each of the years from 1950 to 1956, however, suggest that during this time, at least, there has been little change in any but the oldest age groups where there has been more than a three-fold increase in both sexes (see Table II).

TABLE II

ANNUAL DEATHS PER 100,000 FROM HYPERTENSION, 1950–56

Sex	Age Group (yrs)	1950	1951	1952	1953	1954	1955	1956
Male	30–	1	2	4	4	3	3	4
	35–	7	1	2	5	4	4	10
	40–	13	3	5	5	13	4	9
	45–	5	19	23	9	13	7	10
	50–	29	33	31	35	21	26	23
	55–	63	70	46	46	71	75	66
	60–	81	77	99	78	98	98	102
	65–	109	126	163	110	131	170	120
	70–	179	190	202	168	175	281	220
	75–	186	310	303	303	335	392	424
	80–	219	300	314	382	382	499	438
	85 and Over	201	290	274	328	675	675	639
Female	30–	3	1	2	3	2	2	1
	35–	2	6	2	3	2	5	4
	40–	14	8	8	11	12	7	6
	45–	19	9	20	27	18	14	12
	50–	25	37	46	30	25	27	26
	55–	47	50	39	45	55	53	42
	60–	69	102	86	92	103	92	76
	65–	127	155	122	140	148	153	137
	70–	208	196	245	190	280	267	271
	75–	234	300	360	380	346	474	359
	80–	289	344	400	443	468	573	653
	85 and Over	141	319	282	397	500	794	743

AGE INCIDENCE

Age-specific death rates for cerebrovascular accidents in each of the three decades between 1926 and 1956 are shown in Table III (opposite). When the decades are compared, there has been a tendency, particularly in the post war period, for the death rates to fall in females aged under 54 and to rise in both sexes in the oldest age groups. With the exception of those aged 75 and over in the first decade, and in those aged 85 and over in the other two decades, there is in both sexes a steady upward gradient in death rates for each of the quinquennial age groups in each decade. Table II shows that there is also a steady upward gradient with age for mortality from hypertension.

SEX DIFFERENCES

The sex ratios for each of the three decades are

TABLE III

AVERAGE ANNUAL DEATHS PER 100,000 FROM CEREBROVASCULAR ACCIDENTS

Sex	Age Group (yrs)	Average Annual Death Rate			Indices taking 1926–35 as Unity		Index taking 1935–45 as Unity
Year		1926–35	1936–45	1946–55	1936–45	1946–55	1946–55
Male	35–	9	8	8	0·8	0·8	1·0
	40–	17	16	17	0·9	1·0	1·1
	45–	27	33	26	1·2	0·9	0·8
	50–	56	64	58	1·1	1·0	0·9
	55–	122	102	102	0·8	0·8	1·0
	60–	230	186	190	0·8	0·8	1·0
	65–	281	322	314	1·1	1·1	1·0
	70–	504	647	536	1·3	1·1	0·8
	75–80–	500	744	1,504	1·5	3·0	2·0
	85 and Over	439	760	1,149	1·7	2·6	1·5
Female ..	35–	15	14	11	0·9	0·7	0·7
	40–	29	33	17	1·2	0·6	0·5
	45–	56	54	42	1·0	0·7	0·8
	50–	107	112	89	1·0	0·8	0·8
	55–	168	167	142	1·0	0·8	0·8
	60–	291	293	256	1·0	0·9	0·9
	65–	359	435	379	1·2	1·1	0·9
	70–	533	688	622	1·3	1·2	0·9
	75–80–	532	806	1,595	1·5	3·0	2·0
	85 and Over	437	815	1,226	1·9	2·8	1·5

shown by quinquennial age groups for cerebrovascular accidents in Table IV. At all ages and at all times this has been a commoner certified cause of death in females than in males. The sex difference is most marked in the youngest groups and becomes steadily less until the ratio approaches unity in the oldest groups. There is remarkably little secular trend in the sex ratio at any age group.

Table V shows the sex ratios for hypertension for the period during which comparable statistics are available; with the exception of the youngest groups, where the death rates are very low, and the groups aged 55–64, hypertension also seems to be a commoner certified cause of death in females than in males, but there is no comparable tendency for the ratio to increase with age.

TABLE IV

SEX RATIO (M/F) FOR CEREBROVASCULAR ACCIDENTS IN IRELAND, BY AGE GROUP AND DECADE, 1926–55

Age Group (yrs)	Decade		
	1926–1935	1936–1945	1946–1955
35–	0·6	0·5	0·7
40–	0·6	0·5	1·0
45–	0·5	0·6	0·6
50–	0·5	0·6	0·6
55–	0·7	0·6	0·7
60–	0·8	0·6	0·7
65–	0·8	0·7	0·8
70–	0·9	0·9	0·9
75–80–	0·9	0·9	0·9
85 and Over	1·0	0·9	0·9

TABLE V

SEX RATIO (M/F) FOR HYPERTENSION IN IRELAND, BY AGE GROUP, 1950–56

Age Group (yrs)	1950–1956
30–	1·5
35–	1·7
40–	0·8
45–	0·7
50–	0·9
55–	1·3
60–	1·0
65–	0·9
70–	0·8
75–	0·9
80–	0·8
85 and Over	1·0

Geographical Differences Within the Republic of Ireland

Death rates for the period 1951 to 1955 standardized to the population in 1951 for the counties and county boroughs in the Republic are shown in Table VI and Fig. 2, and in Figs 3 and 4 (opposite).

Table VI

AVERAGE ANNUAL DEATH RATES PER 100,000 (AT AGE 35+) FOR COUNTIES AND BOROUGHS, 1951–1955 STANDARDIZED TO THE POPULATION IN 1951, BY SEX

Cause of Death		Cerebro-vascular Accidents		Hyper-tension	
Sex		Male	Female	Male	Female
Counties	1. Carlow	182	159	83	64
	2. Cavan	182	266	107	126
	3. Clare	144	179	45	49
	4. Cork	192	245	55	69
	5. Donegal	211	225	50	59
	6. Dublin	155	199	87	126
	7. Galway	153	188	82	72
	8. Kerry	147	203	88	89
	9. Kildare	130	216	39	61
	10. Kilkenny	161	194	80	88
	11. Laoghis	191	268	70	89
	12. Leitrim	199	259	61	68
	13. Limerick	124	187	53	82
	14. Longford	117	107	84	61
	15. Louth	184	254	55	48
	16. Mayo	110	132	62	78
	17. Meath	194	243	103	96
	18. Monaghan	157	254	68	94
	19. Offaly	173	243	78	73
	20. Roscommon	142	182	74	91
	21. Sligo	191	192	78	96
	22. Tipperary, N.R.	208	223	74	76
	23. Tipperary, S.R.	218	260	56	86
	24. Waterford	178	290	43	72
	25. Westmeath	185	281	79	74
	26. Wexford	242	303	82	75
	27. Wicklow	307	369	109	94
Boroughs	28. Cork C.B.	224	255	76	80
	29. Dublin C.B.	209	231	120	136
	30. Dun Laoghaire C.B.	286	332	140	135
	31. Limerick C.B.	141	143	60	52
	32. Waterford C.B.	194	275	38	48
Republic of Ireland		182	226	77	90

Although it might be maintained that the urban death rate from cerebrovascular accidents is on the average a little higher than the rural death rate from this cause, and that death rates in the south-east are higher than those in the north-west, there is no very strongly defined geographical pattern, nor is there a very wide range of variation. The highest death rate (369 for women in County Wicklow) is only three and a half times higher than the lowest (107 for women in County Longford). In the case of hypertension, although the rates are on the whole much lower, the range of variation is much the same, the rate of 140 for males in Dun Laoghaire being 3·7 times higher than the rate of 38 for males in Waterford County Borough; but the overall pattern seems to be even less clearly defined.

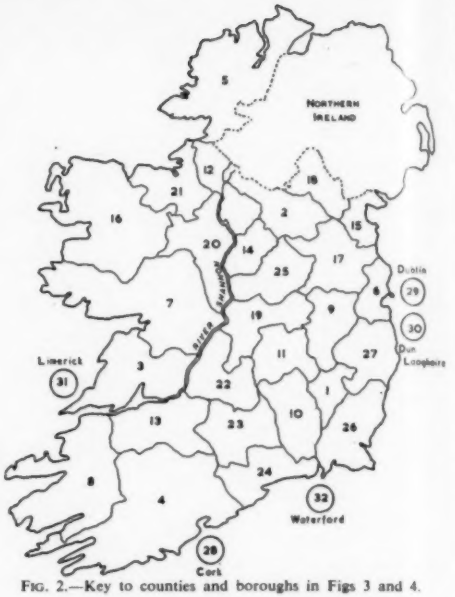

Fig. 2.—Key to counties and boroughs in Figs 3 and 4.

Occupation and Income

Causes of death are not published by trade and occupation in the Republic because the necessary social classification offers considerable technical difficulties. It has not therefore been possible to analyse the present data in terms of social status or occupational hazard.

Comparison of Mortality from Cerebrovascular Accidents and Hypertension in the Republic of Ireland with that in Other Countries

Tables VII and VIII (overleaf) show death rates for the two groups for various countries in 1955, standardized against the Irish population in 1951, the countries being ranked in order of the male death rates. Three aspects of these Tables are worthy of comment; first, the lowest death rate from cerebrovascular accidents is considerably higher than the highest death rate ascribed to hypertension; secondly, although the sex ratio for cerebrovascular accidents does not range appreciably from unity for any of the countries, if the countries had been ranked according to female death rates the order would change a great deal—for example, Sweden would rise from eleventh to fourth position, and France would fall from eighth to fifteenth. In the case of hypertension it is also true that the order of ranking by female death rates would be quite different from that by male death rates, but this is

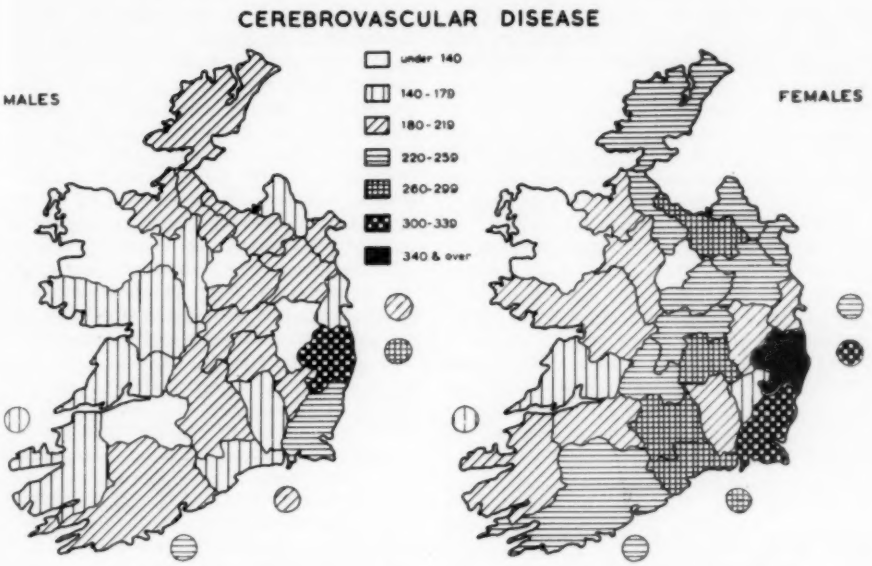

FIG. 3.—Average annual death rates, 1951–1955, from cerebrovascular disease, by counties and boroughs (see also Table VI). The scheme used for shading is the same for both sexes.

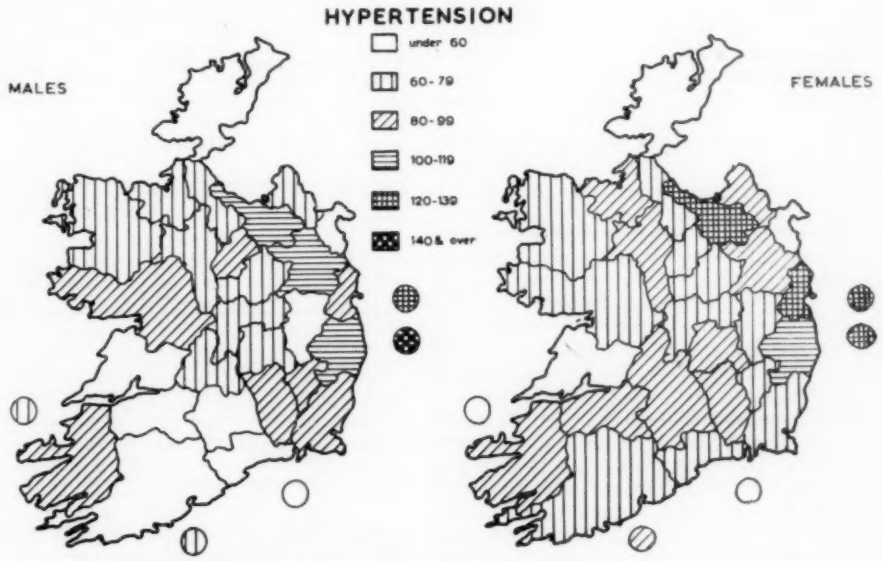

FIG. 4.—Average annual death rates, 1951–1955, from hypertension by counties and boroughs (see also Table VI). The scheme used for shading is the same for both sexes.

less surprising because the sex ratio varies rather more widely than in Table VII and there is very little difference between the male rates for the first nine countries.

TABLE VII

DEATH RATES PER 100,000 FROM CEREBROVASCULAR DISEASE (AT AGE 40+) FOR VARIOUS COUNTRIES IN 1955 (STANDARDIZED TO IRISH POPULATION, 1951)

Country	Sex		Ratio M/F
	Male	Female	
1. Japan	774	611	1·3
2. Finland	476	511	0·9
3. Italy	465	413	1·1
4. Scotland	440	538	0·8
5. England and Wales	421	412	1·0
6. Switzerland	393	402	1·0
7. Australia	378	428	0·9
8. France	378	307	1·2
9. U.S.A.	342	336	1·0
10. Denmark	338	386	0·9
11. Sweden	331	478	0·7
12. Norway	324	371	0·9
13. Netherlands	319	380	0·8
14. Canada	318	343	0·9
15. New Zealand	294	395	0·8
16. Ireland	250	301	0·8

TABLE VIII

DEATH RATES PER 100,000 FROM HYPERTENSION (AT AGE 40+) FOR VARIOUS COUNTRIES IN 1955 (STANDARDIZED TO IRISH POPULATION, 1951)

Country	Sex		Ratio M/F
	Male	Female	
1. England and Wales	128	109	1·2
2. U.S.A.	120	172	0·7
3. Australia	118	125	0·9
4. Ireland	112	120	0·9
5. Scotland	112	107	1·0
6. Finland	104	196	0·5
7. Canada	104	136	0·8
8. New Zealand	101	129	0·8
9. Italy	93	109	0·8
10. Japan	61	53	1·1
11. Norway	60	68	0·9
12. Switzerland	56	81	0·7
13. Sweden	56	76	0·7
14. Denmark	51	77	0·7
15. Netherlands	39	62	0·6
16. France	23	20	1·1

CORRELATION BETWEEN DEATHS FOR CEREBROVASCULAR ACCIDENTS, HYPERTENSION, AND CORONARY ARTERY DISEASE

In order to investigate further the interrelationships between the mortality patterns of the two diseases being studied here and that of coronary artery disease, a correlation analysis was carried out between the standardized death rates shown in Tables VI, VII and VIII above and those in Tables IV and VI in the earlier publication (Acheson and Thornton, 1958). The results are given in Tables IX, X, and XI.

Table IX shows that, despite the variations in sex ratio referred to above, the mortality in the two sexes for each of the three diseases is highly correlated both inside Ireland and in the comparison between Ireland and other countries. The correlations tend to be higher in the international comparison than inside Ireland, but in each case they are highest for coronary artery disease. The cross-correlations between the three diseases inside Ireland are given in Table X which shows that all the correlations for males are higher than those for females; in females the highest correlation is that between coronary artery disease and hypertension, and the male correlation between these two diseases is also high. However, the correlations between the other diseases for both sexes are positive, and (with the exception of those shown between cerebrovascular accidents and hypertension for females) they are significant at the 5 per cent. level at the least.

TABLE IX

CORRELATION BETWEEN MALE AND FEMALE STANDARDIZED DEATH RATES FOR THE 32 IRISH COUNTIES AND FOR THE 16 COUNTRIES SHOWN IN TABLES VII AND VIII

Cause of Death	32 Irish Counties and Boroughs, 1951–1955	16 Countries shown in Tables VII and VIII
Hypertension	+0·75	+0·87
Cerebrovascular Disease	+0·80	+0·79
Coronary Artery Disease	+0·83	+0·94

TABLE X

CORRELATION BETWEEN STANDARDIZED DEATH RATES FROM THREE DISEASES IN IRELAND

Variables	Male	Female
Coronary Artery Disease and Hypertension	+0·57****	+0·42**
Coronary Artery Disease and Cerebrovascular Disease	+0·58****	+0·35*
Cerebrovascular Disease and Hypertension	+0·47***	+0·27

* $P<0·05$ *** $P<0·01$
** $P<0·02$ **** $P<0·001$

TABLE XI

CORRELATION BETWEEN STANDARDIZED DEATH RATES FROM THREE DISEASES IN 16 COUNTRIES (SEE TABLES VII AND VIII)

Variables	Male	Female
Coronary Artery Disease (1954) and Hypertension (1955)	+0·67***	+0·63***
Coronary Artery Disease (1954) and Cerebrovascular Disease (1955)	−0·42	−0·20
Cerebrovascular Disease (1955) and Hypertension (1955)	−0·07	+0·01

*** $P<0·01$

The international analyses (see Table XI) also show that the highest correlation in both sexes is that between coronary artery disease and hypertension. Beyond this, however, there is little further

similarity between Tables X and XI, for the international analysis fails to demonstrate any correlation between cerebrovascular accidents and hypertension for either sex, and such correlation as exists between cerebrovascular disease and coronary artery disease is negative, whereas in Ireland it is positive.

Discussion

Before attempting to evaluate the positive findings made in this study, the shortcomings of the data should be stressed. Cerebrovascular accidents most commonly involve the middle cerebral artery, and result in hemiparesis or hemiplegia. Of all causes of death, apoplexy with hemiplegia is probably the easiest to distinguish. and therefore carries the least observer error in certification. Yet this apparent simplicity in diagnosis is most deceptive because, while the classical apoplexy is usually caused by haemorrhage, the haemorrhage may be into the brain stem, or the lesion may be due to a thrombus of any of a number of arteries. Furthermore, an almost identical clinical picture may develop as a consequence of embolism, a lesion of quite distinct aetiology from haemorrhage or thrombosis. The arrangement of the International List has made it impossible to distinguish between the three, partly because, throughout the period studied, cerebral thrombosis and cerebral embolism have always been grouped together, and partly because between 1926 and 1939 the supplementary heading of hemiplegia was offered. Thus, under the heading of cerebrovascular accidents, we are really considering three distinct diseases which may produce a single clinical pattern; the latter misleadingly obvious to doctor and layman alike.

Hypertension, on the other hand, can be diagnosed with certainty in the home (where most deaths occur) only by the use of a sphygmomanometer while the patient is alive. Very often, moreover, there is a fall in blood pressure in agonal heart failure, so that a doctor attending the last illness may not realize that the blood pressure was previously raised, with the result that death is ascribed to "cardiac failure" or "myocardial degeneration" without any mention of hypertension. Thus, many hundreds of deaths which were in fact due to hypertension must appear among the 4,965 classified as caused by "other myocardial degeneration" (Int. List No. 422) out of a total of 10,902 cardiac deaths in the Republic of Ireland in 1956.

A further difficulty stems from the fact that, even in cases in which the hypertension has been recognized, it may be listed on the death certificate as a contributary cause of death, or as a complication, rather than as the disease which is really responsible for death. In many countries, therefore, it will not appear in the national vital statistics. Broderick (1955), in a detailed analysis of death certificates in the county borough of Dublin, has shown that this is indeed the case. During the year 1952 a total of 310 deaths was certified as due to hypertension (Int. List Nos. 440–447). In a further 386 certificates, however, hypertension appeared as a complication or as a contributary cause of death. In 134 of these cases cerebral haemorrhage (Int. List No. 331) was certified as being the underlying disease directly responsible for death and in 102 cases coronary artery disease (Int. List No. 420). On the one hand, this evidence indicates that the published mortality from hypertension underestimates the prevalence of the disease by a factor of two at least; on the other hand, it amply supports the statement made at the beginning of this paper that cerebral vascular disease and coronary artery disease are commonly complicated by hypertension. Thus, statistics compiled from death certificates will tend to underestimate the prevalence of hypertension.

A further complication is that the degree of underestimation of the prevalence of hypertension will vary from place to place depending, among other things, on the national laws and practices concerning the completion of the death certificate and the coding of the cause of death. These in turn will depend on the medical tradition in the schools where the doctors were trained, on their knowledge of the methods of their national departments of vital statistics, and on their understanding of the importance of imparting accurate standardized information. National differences will depend too upon the extent to which the authorities confine their interest in the death certificate solely to the ruling out of foul play and how much to the compilation of accurate mortality statistics. W.H.O. has attempted to do away with such idiosyncrasies by drawing up a standard form of death certificate and by publishing a manual of rules for abstracting information from certificates on which several causes of death appear (W.H.O., 1948). Both of these are used by many member countries, including Ireland. Other matters of importance are the proportion of the population which dies in hospital, and the amount the doctor certifying the death knows about his patient. It is, therefore, perhaps a little incongruous that the International List should offer six sub-groups for the classification of hypertension which may require the diagnostic facilities of a hospital for their identification, and yet should fail to separate cerebral embolism and thrombosis which can often be distinguished by careful history-taking.

The findings in the present analysis will have to be considered, these shortcomings being borne in mind. If it is accepted that the rise in comparative mortality indices for cerebrovascular disease in the Republic of Ireland in both sexes in 1931, 1940, and 1950 are due to changes in the International List, the most striking characteristic of Figs 1 and 2 is the extraordinary steadiness of these indices between 1926 and 1950. During the final 6 years, however, there can be little doubt that a genuine rise has taken place. Much if not all of this rise is probably due to improvement in the collection of statistics. During this time there has been a drive in the more rural and isolated parts of the Republic to ensure that all deaths are properly certified, with the result that there has been a very sharp fall in the numbers of deaths ascribed to senility or remaining uncertified (for further details see Acheson and Thornton, 1958). This trend is also reflected in Tables II and III, where the apparent rise in death rates from hypertension as well as from cerebrovascular disease among the older age groups must also be largely, if not entirely, due to more accurate certification.

The secular fall in the death rate from cerebrovascular accidents in women aged 35–54 is more likely to be real. One possible cause of this is the decrease in the number of cases of rheumatic heart disease, a decrease which is reflected in the fact that the death rates from this cause in Ireland have been almost halved during the 30 years under consideration. Therefore, deaths from cerebral embolism have presumably fallen too, because cerebral embolism is very often associated with rheumatic carditis, and is more likely to develop in young women than in old people of either sex. The available statistics are not, however, suitable for examining this hypothesis in any detail.

The most interesting aspect of the geographical data is the correlation analysis shown in Tables IX to XI. Evidently there is no great discrepancy in Ireland, or in the other countries studied, between death rates in the two sexes, for the between sex correlations vary from 0·75 to 0·94. The value of 0·94 for coronary artery disease can be interpreted as giving indirect supportive evidence for the conclusion, drawn from the Irish comparative mortality indices (Acheson and Thornton, 1958), that the rate of increase in female death rates from this disease has been as rapid as that in male death rates. It can also be looked upon as a reflection of the accuracy of diagnosis, however, for Tables X and XI show that the only consistently high correlations are those between coronary artery disease and hypertension. It could well be argued that a similar type of *modus vivendi* or environment is a contributory factor to causing death from the two diseases. On the other hand, these diseases require refined medical techniques for their diagnosis, and the observed correlation may really reflect the standard of medical care. In the previous paper (Acheson and Thornton, 1958), it was concluded that the increase in mortality from coronary artery disease was not entirely attributable to *improved* diagnostic methods. This is not to say, however, that the level of medical skill in a community has no bearing on the certified death rate from coronary artery disease; it certainly must have some bearing, as indeed it must on the certified death rate from hypertension. Furthermore, as Tables VI, VII, and VIII show, the geographical areas both inside and outside Ireland with the highest mortality from these causes tend to be those with the most highly developed medical services; thus, although the social culture which Morris (1955) has dubbed the "Western" way of life may cause people to die from hypertension, as it is known to cause them to die from coronary artery disease, the statistical relationship found between the death rates from the two causes in the present study is more than likely to be also a reflection of diagnosis and certification.

There is a wide difference in the correlation between cerebrovascular disease and hypertension or coronary disease shown in Table X as compared with that shown in Table XI. In Ireland the areas with high death rates for one of the three certified causes of death tend to have high death rates for the other two, particularly in males. In the international comparison this is clearly not the case, the most striking difference being that Japanese males show the highest standardized death rate for cerebrovascular disease and the lowest for coronary artery disease (see Acheson and Thornton, 1958). While such discrepancies between one country and another may to some extent be due to true differences in the epidemiology of disease, variations in diagnostic and statistical technique cannot be ruled out as an important contributary factor.

Few facts of epidemiological value have resulted from this analysis. It does, however, illustrate admirably the pressing need (stated by W.H.O., 1959) for a thorough scientific study of the methods whereby vital statistics are collected all over the world.

Summary

(1) Between 1926 and 1956 comparative mortality indices for both sexes in the Republic of Ireland show a rise for cerebrovascular accidents. This is probably entirely due to changes in the International Classification of Causes of Death and to more

accurate certification of death. A similar study of the secular trend in mortality from hypertension was impossible because of alterations in the International List; between 1950 and 1956 however, there was no suggestion of consistent change, except in the oldest groups where death rates rose but certification has improved.

(2) Both cerebrovascular accidents and hypertention were more commonly certified as causing death in women than in men.

(3) In respect of cerebrovascular disease, the average standardized death rates for the period 1950–1955 tended to be higher in urban than in rural districts; they also tended to be higher in the counties of the eastern seaboard than elsewhere in the rural parts of the Republic. For hypertension the average standardized death rates for the conurbation of Dublin were higher than those elsewhere in the Republic; otherwise there was little consistent geographical pattern.

(4) Of sixteen countries whose vital statistics are regularly published by W.H.O., Ireland in 1955 had the fourth highest male death rate from hypertension but the lowest from cerebrovascular accidents—all death rates being standardized to the Irish population in 1951.

(5) A correlation analysis was carried out between death rates for hypertension, cerebrovascular accidents, and coronary artery disease for the 32 administrative areas inside Ireland and for Ireland and the other fifteen countries, with the following results:

(*a*) There were high positive correlations between the sexes for all three diseases, the highest correlation in each analysis being that for coronary heart disease (+0·83 in Ireland and +0·94 in the International comparison).

(*b*) There was a highly significant correlation between hypertension and coronary artery disease both inside Ireland and internationally.

(*c*) Inside Ireland the other correlations were positive, and the only one which was not significant was that between cerebrovascular accidents and hypertension for females.

(*d*) In the international comparison, correlations between cerebrovascular accidents and hypertension differed little from zero, and those between coronary artery disease and cerebrovascular disease were negative.

(6) In considering the possible significance of these findings, the grave shortcomings of mortality figures for cerebrovascular accidents and hypertension as a basis for epidemiological studies are stressed.

I am most grateful to Miss Marie Geoghegan who bore the brunt of the computing; to Dr. Donal MacCarthy and Mr. J. F. Knaggs of the Central Statistics Office, Dublin for making available unpublished data; and to Prof. W. J. E. Jessop for encouragement. Prof. W. S. Walton kindly read the text. The research was supported by a grant from the Medical Research Council of Ireland.

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THE SOCIETY FOR SOCIAL MEDICINE

A meeting will be held at Oxford on September 29 to October 1, 1960, at St. Hugh's College. The fourth annual general meeting will be held at 2 p.m. on September 29, and the Scientific Programme will be as follows:

THURSDAY, SEPTEMBER 29

3.0 p.m. SYMPOSIUM: EPIDEMIOLOGY OF LEUKAEMIA

Opening Speakers:

(1) Children - *Alice Stewart*
(2) Adolescents - *J. A. Lee*
(3) Adults - *F. W. Gunz*

FRIDAY, SEPTEMBER 30

9.15 a.m. Distribution of Multiple Sclerosis in U.S.A. Veterans — *E. D. Acheson*

9.45 a.m. Diabetes and Epilepsies in a Scottish Community — *G. D. Forwell* and D. Cameron

10.15 a.m. Social Class Mobility in Chronic Bronchitis — *Susan Meadows*

11.15 a.m. Mortality among Gas Retort-House Workers — *R. Doll* and R. Fisher

11.45 a.m. Bus-Driver Accidents in Northern Ireland — *P. Froggatt*

12.15 p.m. Maternal Physique and Toxaemia of Pregnancy — *R. Lowe*

2.30 p.m. The Mechanism of Rhesus Iso-immunization — *E. G. Knox*

3.0 p.m. Differences between Populations in respect of the Tertiary Sex-ratio — *F. A. E. Crew*

3.30 p.m. Genetic Factors governing the Shape of the Hand — *D. Hewitt*

4.30 p.m. The Influence of Some Environmental Variables on the Incidence of Anencephalus — *R. G. Record*

5.0 p.m. Birth Rank and Family Size as Factors in Educational Selection — *J. W. B. Douglas*

SATURDAY, OCTOBER 1

9.15 a.m. Psychiatric Illness in Adolescents after Six Years — *Lilli Stein* and W. Warren

9.45 a.m. Institutionalism in Mental Hospitals — *J. K. Wing*

10.15 a.m. Recent Admissions to Mental Hospitals in Salford — M. Susser and *Zena Stein*

11.15 a.m. Changes in Family Size — *J. A. H. Waterhouse*

11.45 a.m. The Quality of Medical Care — *R. F. L. Logan* and G. Forsyth

12.15 p.m. Late Effects of Breast-feeding — *Alice Stewart*

Speakers' names in italics